"It is wrong to trust the natural body to empirics, who commonly have a few receipts whereon they rely, but who know neither the causes of diseases, nor the constitutions of patients, nor the danger of accidents, nor the true methods of cure."

FRANCIS BACON (1561–1626).

DISEASES

OF THE

SOFT STRUCTURES OF THE TEETH AND THEIR TREATMENT

A TEXT-BOOK FOR STUDENTS AND PRACTITIONERS

BY

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PREFACE.

In the following pages an effort is made to present to the student and practitioner of conservative dentistry a comprehensive study of the treatment of diseases of the soft structures of the teeth, based upon their clinical pathology. The difficulties which presented themselves to the author in systematizing the subject were the many conflicting statements as found in the literature. An attempt has been made to avoid vague information and to elucidate only clinical facts as related to the pathology of each individual disease and the subsequent application of rational therapeutic measures for its treatment.

The first part of the book comprises the diseases of the dental pulp, while the second part will treat of the diseases of the pericementum including pyorrhea alveolaris.

It is to be hoped that the second part will be available in the early spring of 1923.

H. P.

Evans' Institute,
September, 1922.

CONTENTS.

DISEASES OF THE SOFT STRUCTURES OF THE TEETH AND THEIR TREATMENT.

INTRODUCTION.

The historical evolution of the practice of the healing art distinctly indicates that it has always been governed by the ruling notions of general pathology. The early fathers of medicine regarded the body in its physiologic concept as being composed of the four elements of the philosopher Empedocles (490–430 B.C.), *i. e.*, fire, water, air and earth. According to the latter's conception all material substances in the world were composed of these four elements. Basing their ideas of health and disease upon this philosophic doctrine, the ancient pathologists supposed that the body contained as an inherent property these four cardinal elements in the form of juices—the humors, *i. e.*, blood (*sanguis*) phlegm (*phlegmon*), yellow bile (*choler*) and black bile (*melancholor*). It was assumed that life depended upon a proper mixture of these humors which constituted its well-being known as *eucrasia*. Whenever the normal equilibrium of the humoral mixture became disturbed, disease—*dyscrasia*—resulted producing abnormal temperaments—*complexiones*—such as heat, moisture, dryness and cold, which in turn corresponded to the properties of the four original elements. As a consequence, this humoral conception of the nature of disease which was principally fostered by Hippocrates (460–377 B.C.) is referred to by the medical historian as humoral pathology, and its predominating influence was recognized as late as the middle of the Fifteenth Century. A strong impetus was given to it by the famous Greco-Roman physician, Galen (130–201). He considered an abnormal condition of the blood as the primary cause of disease; to him any deviation from the normal indicated putrefaction and, therefore, his therapy centered about the administration of drugs which caused elimination, *i. e.*, cathartics, and in the regulation of the diet. The dogmatic influence of galenic therapy which held undisputed sway for more than fourteen centuries received a severe check by the introduction of chemistry into clinical practice by Paracelsus (1493–1541). Paracelsus taught doctrines essentially his own and strongly condemned the existing

methods of teaching. He denounced the custom of blindly following the teachings of Celsus, Galen, Avicenna and other orthodox writers and, on the feast of St. John, 1527, the students having lit a bonfire in front of the University of Basel, Paracelsus seized the opportunity to throw a copy of Avicenna's *Canon*—in those days the leading work on the practice of medicine—into the flames, saying: "Into St. John's fire, so that all misery may go into the air with the smoke." He conceived the idea that disease was the result of abnormal manifestation of life. The spiritual principle of life—*archæus*—is bound up with the body as a whole and consists of three elements: sulphur, mercury and salt. However, he used these terms in a purely symbolic manner. When an organic substance burns, that part which is destroyed by fire is sulphur, that which volatilizes and rises as smoke is mercury, and the ash is salt. The resulting slag settles in the various parts of the body like the tartar in the wine cask and causes tartaric diseases. The body juices become altered and blood poisoning—*acrimonia*—results and consequently, only physiologic therapeutics must be employed to reconstruct the dearranged system. In disease the archæus has to bear the brunt of the burden, consequently Paracelsus' iatrochemical therapeutics centered about the reconstruction of this spiritual principle which is only possible by employing the spiritual force of medicinal substances—*quinta essentia*—which he isolated and dispensed in the form of tinctures and extracts—*arcana*. These arcana as obtained from plants and minerals must bear a certain specific resemblance or "signature" to the disease and are utilized as alteratives which so favorably modify nutrition as to overcome morbid processes. Sylvius (1614–1672) was one of the strongest exponents of the theories of Paracelsus and materially assisted in the popularization of his teachings in France. The discovery of the circulation of the blood by Harvey (1578–1658) and the introduction of scientific methods in the study of morbid anatomy by Morgagni (1682-1771) which bore most excellent results in the hands of John Hunter (1720–1793) and Bichat (1771–1802) laid the foundation for the more ready comprehension of the nature and causes of diseases. Andral (1787–1876), of Paris, and Rokitansky (1804–1878), of Vienna, were the principal modern exponents of humoral pathology.

The teachings of humoral pathology were by no means universally accepted. Almost simultaneously with its inception we find the creation of a contemporary school which based its conception of disease upon the composition of solid natural organic and inorganic substances as conceived by the Greek philosopher Democrites (460 B.C.), *i. e.*, the existence of atoms. His doctrine centered about the hypothesis that all material substances were composed of these atoms, and, as a consequence, the density of a mass depended upon the relationship of the spaces between their surfaces which are

filled with air. Health—*status strictus*—is an expression of the normal equilibrium of the affinity between atoms and space while disease—*status laxus*—indicates a greater or lesser density of the relative space occupied by the atoms. Medical historians refer to Democrites' conception of disease as solidary pathology; in general, it has received only meager support.

Stahl (1660–1734) assumed that disease was an expression of the dearrangement of the immortal soul which controls all functions of the living body. This vitalistic pathology found its most ardent advocate in the then flourishing medical school of Montpellier (1700–1800) of southern France, and its teachings reached far into the nineteenth century. The well-kno vn physiologist Johannes Mueller (1801–1858) was its last prominent exponent.

Physiologists had observed that the soft tissues of the body, the muscles, were possessed of peculiar vital phenomena, which distinctly differentiated living from dead tissues. It was left to Albrecht von Haller (1708–1777) to show that electric stimulation would cause contraction of the muscles and that this phenomenon was an expression of the increased sensibility of an irritated nerve. These observations had a most profound influence upon the conception of disease and in the annals of medical history it is recorded as neuropathology.

Humoral pathology and so-called rationalism in therapeutic procedures were the two predominating factors which governed the practice of medicine and incidentally of dentistry at the beginning of the eighteenth century. This supposed rationalism started almost simultaneously in various parts of Europe. Sydenham (1624–1689), of London, Boerhaave (1668–1738), of Leyden, Van Swieten (1700–1772), of Vienna, Hoffmann (1660–1742), of Halle, and Stahl (1660–1734), of Berlin, were the most influential exponents. The growing tendency of overdrugging received a healthy check through the introduction of Hahnemann's method (1810) of treating disease with very small doses and single remedies, which, combined with other extreme changes in therapeutics, resulted in the foundation of the homeopathic school. No definite knowledge regarding drug action had become available to the practising physician, and as a consequence of the empiric administration of drugs, it became quite customary to poke fun at those who regarded drugs necessary in the treatment of diseases. Especially Skoda and Dietl (1830–1870), of the Vienna school, expressed erratic views in regard to drug medication, and both extremists were wont to express their ill-founded skepticism by stating that: There are no real therapeutists—there are only lucky physicians. Bearing in mind the fact that no tangible knowledge of pharmacology existed at that time, our judgment of these outbursts of overzealous critics is materially modified when we consider that even at this day the

drugless "Christian Scientist" and the supporter of the "Emmanuel movement" hold sway in the minds of the credulous.

About the middle of the last century a complete change in the conception of the causes of disease and the subsequent administration of remedial measures for their eradication occurred, which must be primarily credited to the ingenuity of the master-mind of Rudolph Virchow (1821–1902). His name will be forever indelibly engraved upon the annals of medical lore. The ready comprehension of the present possibilities within the domain of clinical pathology are made possible only by an intelligent study of his doctrine. The revolutionary change took place with the publication of his work, *Cellular Pathology*, in 1858. "These lectures," as he stated in the preface of this classic in modern medicine, "were particularly intended as an attempt to offer, in a better arranged form than had hitherto been done, a view of the cellular nature of all vital processes, both physiologic and pathologic, animal and vegetable, so as distinctly to set forth what even the people have long been conscious of—namely, the unity of life in all organized beings, in opposition to the one-sided humoral and neuristical tendencies which have been transmitted from the mythical days of antiquity to our own times, and at the same time to contrast with the equally one-sided interpretations of a grossly mechanical and chemical bias—the more delicate mechanism and chemistry of the cells."

Virchow's cellular pathology was founded upon certain observations made by the anatomist Bichat, the originator of histologic research, who stated that life depended upon the physiologic interaction of organic structures. Schleiden (1804–1881), the botanist, and Schwann (1810–1882), the physiologist, had shown that all living tissues are composed of minute cells and Remak (1852) had formulated a law regarding the relationship of these cells to embryonic life by stating: *Omnis cellula e cellula*. It is the recognition of this principle of cell begets cell, that enabled Virchow to construct his epoch-making concept of pathology which, in reality, is merely a deviated physiology.

The doctrine of cellular pathology as enunciated by Virchow is very slowly but steadily recognized within the specialized field of dental medicine and surgery. Such names as Wedl, Tomes, Saunders, Abbott, Magitot, Metnitz, Barrett, Burchard, Miller, Boedecker, Black and many others who are still at work and too numerous to mention will forever be recognized as leaders who have been or are still actively engaged in assisting in the slow metamorphosis which by necessity, dentistry has to undergo to entitle it to the cognomen of a learned profession.

Medicine is sometimes considered a science and sometimes an art. Both conceptions are correct; it depends merely upon the viewpoint from which the subject is approached. Medicine is a

science by its means of study and an art by its application. The object of medical science is to study disease; the aim of medical art is to cure disease, to relieve suffering and to maintain health. In general, it may be stated that the science of medicine is concerned with the study of the fundamental problems of biology as pertaining thereto, while the art of medicine treats of the study of disease, *i. e.*, pathology, and the means and methods employed in relieving the sick and in favorably modifying the evolution of disease, *i. e.*, therapeutics. The teaching of medical art as it is carried out on the sick constitutes *clinics*.

Pathology, as stated above, is that part of medical science which embraces the study of diseases in all its aspects and explains the origin, cause, clinical history and nature of the various morbid conditions which may disturb the economy and, consequently, it is the composite of numerous subdivisions.

Etiology is concerned with the study of the causes of diseases. Morbific causes may be internal or intrinsic, external or extrinsic, ordinary, specific, primary or secondary and exciting. These causes may also be classified as being predisposing or determining. Predisposing causes may prepare the body or a part thereof by rendering it weak and less resistant, while the latter are the immediate or specific causes of disease. *Pathogeny* tries to explain by what mechanism these causes act on the living organism by disturbing the state of its health and by abolishing its resistance. *Morbid* or *pathologic anatomy* discusses the changes of the tissues after death and *pathologic physiology* portrays the morbid reaction of the living organisms from pathogenic causes. *Symptomatology* furnishes an account of the signs and symptoms appreciable during life, while *nosology* describes and classifies disease. *Symptoms* may be objective or subjective; objective when evident to the senses of the observer, as redness, swelling, high temperature, etc.; subjective when felt or known only by the patient, as pain, nausea, etc. The *prodromes* are the earliest recognizable symptoms, as rigors and chills during the invasion of fever while *sequels* are morbid phenomena which are left as a result of disease. *Syndromes* represent the aggregate of the symptoms associated with any morbid condition, and together they constitute the picture of the disease. An *acute* disease is one in which the invasion is sudden and rapid and, as a rule, severe; when the symptoms develop less rapidly and are less intense the disease is said to be *subacute*. If the disease is very slow in unfolding its salient features with an obscure blending of the various symptoms it is said to be *chronic*.

Diagnosis, or discrimination of the disease, determines the place which it occupies in nosology and *prognosis* tries to fortell its evolution. A complete diagnosis includes: (1) The naming of the disease; (2) the recognition of its state, peculiarities and compli-

cations; and (3) the estimation of dangers existing or liable to arise. In a *direct diagnosis* the morbid condition is revealed by a combination of clinical phenomena or by symptoms belonging to a particular disease and hence they are termed pathognomonic symptoms. A *differential diagnosis* results from the elimination of diseases resembling each other, while a *diagnosis by exclusion* proves the absence of all diseases which might give rise to the symptoms observed, with the exception of one, the presence of which is not actually indicated by any positive signs. The diagnosis is the most important and, incidentally, the most difficult part of the art of medicine. It reflects all the various steps involved in the study of disease and in the correlated sciences. The use of the microscope, instruments of precision, chemical and bacteriologic tests, roentgenograms and numerous other adjuncts have been utilized in the construction of the foundation upon which a large part of our knowledge of pathologic processes is built and by it and through it diagnosis has been placed upon a rational basis. The introduction of the electric current as a diagnostic aid in the recognition of diseases of the pulp should be considered as one of the most important advances in the practice of dental therapeutics.

The ultimate object of the clinical practice of medicine is to be able to cure, to relieve and to prevent disease, *i. e.*, the application of therapeutic measures. *Therapeutics* does not consist solely of the administration of drugs, but it requires a comprehensive knowledge of surgery, prophylaxis and dietetics. The application of remedial substances in the treatment of diseased conditions is based on a knowledge of *pharmacology*, and at present it is referred to as *pharmacotherapy*, while *surgery* is that branch of therapeutics which endeavors to cure by manual procedures. *Prophylaxis*, whose principal part is represented by hygiene, dictates the precepts to be followed in avoiding disease and *dietetics* indicates the diet conducive to the restoration and preservation of health.

Nomenclature, *i. e.*, the naming of diseases, is an important division of general pathology. Names of diseases, like names of other things, have originated in a variety of ways and have undergone many changes at the hands of the generations who have suffered from diseases. Some, like Barlow's disease or Bright's disease, have received the names of their supposed discoverer, while others, like paralysis agitans are called for some pronounced sign or symptom. Epulis, as its etymology indicates, was employed by Galen to designate a tumor growing upon the gum, while parulis (growing beside the gum) was used by him to signify a gum boil (chronic alveolar abscess). Again, the term malaria (bad air, referring to the theory of the miasmatic origin of the disease) was used to refer to some apparently causal condition. A few names have had a more sentimental origin—for instance, syphilis, which

gets its euphonious title by way of a poem of a sixteenth century physician, named Fracastorius. A shepherd, named Syphilis, was stricken with the disease by Apollo, in punishment for paying divine homage to the king instead of to the god. The disease stuck to the shepherd and somehow the shepherd's name became firmly attached to the malady.

The real value of medical nomenclature consists in choosing a name that shall suitably express the involved morbid condition, and, if possible, its location. Unfortunately, this is not always possible, since numerous terms by virtue of their antiquity are still retained, as carcinoma, sarcoma, etc.

Inflammation.—The significance of inflammation is usually expressed by adding the suffix *itis* to the name of the anatomical structure involved, thus if the disease be an inflammation of the pulp, it is termed *pulpitis.*

Pain without Inflammation.—The significance of pain *without* inflammation is usually expressed by adding the suffix *algia* to the name of the anatomic structure involved. Thus, if the disease be a morbid condition of the nerve tissue without inflammation, it is termed *neuralgia.* Occasionally, the suffix *dynia* is employed to describe a similar condition. Thus, pain in the tongue without inflammation is referred to as *glossodynia.*

Catarrh.—The significance of a morbid condition indicating the formation of an inflammatory transudate escaping to a mucous surface, known as catarrh, is usually expressed by adding the suffix *rhea* to the name of the transudate or flux. Thus, if the disease be a morbid condition of the alveolus of a tooth associated with a flow of pus it is termed *pyorrhea alveolaris.*

Dropsy.—The significance of an excessive accumulation of watery liquid in a tissue or cavity of the body is usually expressed by adding the prefix *hydro* to the anatomic name of the involved structure. Thus, if the disease be an accumulation of a dropsical transudate within the maxillary antrum it is termed *hydrops antri Highmori.*

Blood Diseases.—To signify a morbid condition of the blood the suffix *emia* is usually employed. Thus, if the disease be an impoverishment of the blood it is termed *anemia;* if blood contains an accumulation of *urea* the disease is designated as *uremia;* a putrid infection of the blood is designated as *septicemia;* a purulent infection as *pyemia,* etc. The significance of a flow of blood, hemorrhage, from an anatomic structure is usually expressed by adding the suffix *rhagia* to the name of the involved tissue. Thus, if a hemorrhage occurs from the small intestines it is termed *enterorrhagia.*

Changes in the Urine.—The significance of alteration in the urine is usually expressed by adding the suffix *uria* to the noun indicating the change. Thus, the term *albuminuria* indicates the presence of

albumin in the urine; *hematuria*, when blood is present in the urine, etc.

Tumors.—The suffix *oma* signifies a tumor, as *osteoma*, a bone tumor; *fibroma*, a fibrous tumor, etc.

The termination of diseased conditions may occur in one of three ways, *i. e.*, cure, secondary processes or death. A cure may manifest itself as *lysis*, *i. e.*, a slow return to health or as *crisis*, *i. e.*, an abrupt change for the better. Secondary processes are those in which the diseased condition is substituted by a morbid process, as infection of the pericementum followed by a tonsillitis. *Metastasis* indicates a shifting of a disease or its local manifestations from one part of the body to another. The complete cessation of all tissue changes is known as *death*. It may manifest itself as an ever increasing debility, *asthenia* (cancer, Bright's disease); as an insufficient quantity or quality of blood, *anemia;* as a non-aëration of the blood, *apnea* (croup); or as death beginning at the brain, *coma* (uremia, narcotic poisoning).

Health and disease (ease and *dis*-ease) are relative concepts concurrently employed to designate states which differ in their manifestations without creating a sharp line of demarcation. Perfect health does not exist; the living organism is always in a state of unstable equilibrium which finds its cause and explanation in the very conditions of life. Living matter does not depend upon a vital force, but it should be considered as reactions produced by the variations of external stimuli.

Numerous colloquial metaphors are utilized to define health and disease. For instance, a clergyman has referred to it as: Health is harmony, disease discord. Roger (1898) defines disease as being the ensemble of the phenomena which are produced in an organism undergoing the action of a morbific cause and reacting against it. Adami (1908) refers to disease as being the expression of a reaction on the part of the cells to injurious agencies, while someone else has stated that disease is a morbid process considered in its entire evolution, *i. e.*, from its initial cause to its final consequence. In short, disease may be defined as expressing the morbid changes which occur when normal tissues react to abnormal influences, thus constituting a definite entity.

It is generally conceded at present that disease may be produced by mechanical, physical, chemical, electric and animate causes and that their initial impulses must be looked for outside of the organism. The lesions as they are produced by these causes do not necessarily remain local; secondary morbid manifestations are most frequently observed as direct sequences. Local causes are usually acting upon a limited point of the organism, while general causes may act on the entire economy or rather on numerous points thereof. Local causes may produce their distinct phenomena at

the point of application or they may give rise to disturbances in distant parts of the body.

The intelligent application of rational therapeutic measures in the treatment of diseases necessitates a comprehensive realization of the gradual development of its underlying phases, *i. e.*, clinical pathology. In the following pages an attempt is made to harmonize the broad principles of the practice of general medicine with that of the specialized field of dental surgery.

In his legal qualifications the dentist is regarded as practising within the compass of a specialized department of the science and art of healing, therefore, it behooves him to thoroughly familiarize himself with the general principles underlying the practice of medicine. Hence, in presenting a discussion of the various dental ailments the writer, in conformity with long-established customs of the practice of the medical art, has adopted the following outline setting forth the salient features in their routine sequences and presenting to the student a complete picture of the respective disease as a whole, relatively speaking, by a detailed interpretation of the clinical aspects of each subject.

Outline of Dental Diseases.

Name of the disease and its synonyms.
Definition of the disease.
Etiology of the disease.
Clinical pathology of the disease.
Symptoms of the disease.
Complications and *sequels* of the disease.
Diagnosis of the disease.
Prognosis of the disease.
Treatment of the disease.

PART I.

DISEASES OF THE DENTAL PULP.

CHAPTER I.

ODONTALGIA.

Odontalgia, toothache, as its name indicates, is, in the majority of cases, the most pronounced symptom of an existing dental disturbance. Toothache, in most instances, is the prime motive which induces the patient to seek the services of the dentist. The relief of the painful condition is his principal desire; to him the conservation of the tooth is, in many instances, a secondary consideration. It is the paramount duty of the practitioner to ascertain, if possible, the cause of the existing pain. He should look upon the aching tooth and its surrounding structure from a biologic point of view, as pain is a manifestation of a vital reaction and, consequently, he should draw upon all the facilities which are offered to him through his knowledge of the medical art so as to be able to institute correct therapeutic procedures for the alleviation of the symptoms of the existing disease, and, if possible, for the eradication of its cause.

Odontalgia is not a disease; it is the subjective painful expression of some pathologic disturbance within the tooth or its surrounding structures. Its etiologic factors may be classified as local, or primary, general, or secondary, and as reflex disturbances. In the majority of cases it will be observed that toothache is the sequence of an inflammatory disturbance within the tooth, *i. e.*, its pulp, or within its external retentive structure, *i. e.*, pericementum. These two important centers of sensation usually may be distinctly differentiated in regard to the subjective manifestations of pain and in regard to the objective symptoms of pathologic alterations. A distinct and clear line of demarcation between the two clinical pictures, *i. e.*, pulpitis and pericementitis, relative to a differential diagnosis should, in the present light of pathologic knowledge, offer no difficulties. As a third group, which, however, quantitatively, is by far overshadowed by the first two groups, we

encounter the reflex odontalgias. While this type of toothache, as stated, is of a comparative rare occurrence, it is, nevertheless, usually very severe in its manifestations and, incidentally, most difficult to diagnose. A detailed discussion of the known facts are, therefore, of paramount importance to the clinical practitioner.

The various causes of the diseases of the pulp and the pericementum, their symptoms, clinical pathology and treatment are discussed in detail in the succeeding pages. Odontalgia at this moment is merely recorded as a clinical entity so as to present to the reader a comprehensive summary of its manifested symptomatology.

The causes of odontalgia may be summarized as follows:

Hypersensitive Dentin.—It may be defined as a state in which the exposed dentin of a vital tooth is painfully responsive to any type of irritation. (Clinical pathology and treatment, see p. 87, *et al.*)

Pulpitis.—Inflammation of the exposed or unexposed dental pulp in its numerous modifications may be caused primarily by mechanical, chemical, thermal, electric or parasitic irritation. Secondarily, it may have its origin in the disturbances of the pericementum, or it may be the sequence of some general disease. (Clinical pathology and treatment, see p. 110 *et al.*)

Secondary Pulpitis.—Secondary manifestations of pain within the pulp of a tooth are of very common occurrence; they may be caused by a primary existing local disturbance within the body of the dental pulp or by a general disease. In the majority of cases of secondary pulpitis the causative agent is a direct or an indirect infection, although general diseases of a non-infectious type, such as a leukemia, locomotor ataxia, anemia, chlorosis, increased intradental blood-pressure and various neuropathic conditions, as hysteria, neurasthenia, etc., may be the exciting factors. (Clinical pathology and treatment, see p. 124, *et al.*)

Pericementitis.—Inflammation of the pericementum may be caused by any type of direct irritation or, secondarily, by local disturbances, *i. e.*, pulpitis or gingivitis, or as a sequence of some general diseases. (Clinical pathology and treatment, see p. 300, *et al.*)

Hyperplasia of Cementum.—Pathologic overgrowth of cementum, frequently referred to as exostosis or hypercementosis, is usually more often found about the roots of the upper teeth. Externally, usually nothing can be seen which would furnish a clue to its diagnosis; only in cases of severe enlargement of the cementum bulging of the gum tissue over the affected tooth may be observed. The pain is usually very indefinite, of a dull, continuous, neuralgiform character and not localized. Occasionally, it may be very excruciating or it may manifest itself as a gnawing paroxysm recurring at irregular intervals. Thermal changes and percussion are negative. Hyperplasia of cementum should be looked upon as a

product of defense instituted by the cementum to protect itself against chronic irritation. It is the analogue of the formation of adventitious dentin in the pulp. The diagnosis is very readily made from a roentgenogram. Treatment: Removal of the tooth or amputation of the affected root.

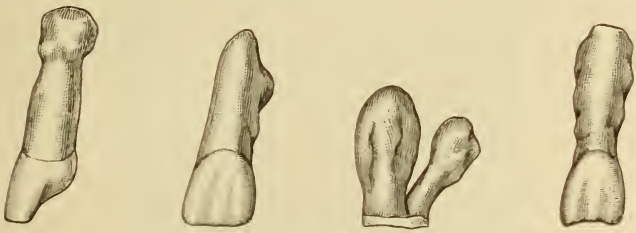


FIG. 1.—Hyperplasia of cementum. (Burchard.)

Disturbed Articulation.—Faulty restoration of a defect of the crown of a tooth by operative procedures may be its direct cause or, if for any other reason a tooth receives a greater impact during closure of the jaws than normal, a painful traumatic pericementitis may result. Treatment: Restoration of normal articulation.

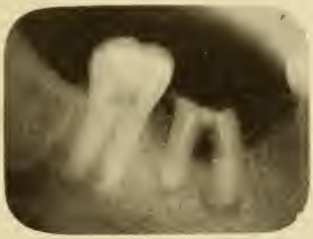


FIG. 2.—Hyperplasia of cementum.

Impacted Teeth.—Impacted teeth usually do not cause painful symptoms unless they meet on their path of retarded eruption an obstruction or they exert pressure upon nerve fibers. The painful symptoms may be of a neuralgiform character or they may be manifestations of a traumatic pericementitis. Occasionally, epileptiform attacks may be observed. The lower third molars, and less so, the upper third molars are most often found to be hampered on their ways through the alveolar process. The "Ahasuerus" among the permanent teeth is the upper canine. In its vicarious excursions it may erupt anywhere about the maxilla and at any age. Although fully developed, the tooth may lie dormant within the body of the jaw for years, or, when irritated by pressure or otherwise, may at any time start on its erratic journey to the periphery. The writer has had occasion to remove impacted canines from every

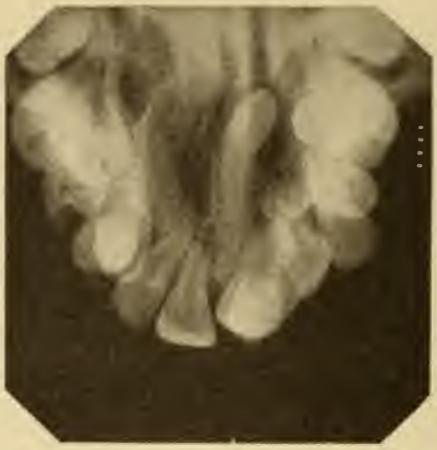


FIG. 3.—Impacted and malformed second incisor.

FIG. 4.—Impacted lower third molar.

FIG. 5.—Impacted upper canines.

available surface of the hard palate, from the nasal cavity and from the maxillary sinus. In one case the vagrant made its appearance at the age of sixty-five in an edentulous jaw over which a plate had been worn for more than thirty years. Impacted teeth frequently show marked absorption lacunæ; genuine caries, however, has probably never been observed.

Root remnants left in the jaws and fractured teeth have been very frequently obstinate sources of most obscure pain. Usually, an existing fistula will assist in locating these broken roots; on rare occasions the fistula may be absent. A roentgenogram will clarify the diagnosis in both instances. Treatment: Removal of the obstruction or of the impacted tooth or root remnant.

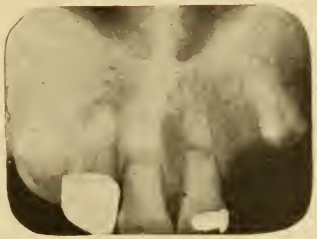


Fig. 6.—Fractured upper incisors.

Overstrain of Single Teeth.—Single teeth standing in a broken arch frequently are overstrained during the process of mastication. If the antagonists of the tooth are missing the tooth may elongate in its socket and thereby expose its root or roots or it may cause traumatic injury of the opposing alveolar ridge. The continuous impact on these teeth during mastication may cause inflammation of the pericementum with all its sequences, while its exposed roots, being denuded of the protective alveolar process and cementum, becomes frequently most excruciatingly hypersensitive. Treatment: Prosthetic replacement of the lost teeth or extraction. (The treatment of hypersensitive dentin is discussed on p. 87, *et al.*)

"Setting Teeth on Edge."—This popular expression as employed by the laity denotes a state of irritation of the pulp brought about by the action of organic acids taken as condiments or being present in foodstuffs upon denuded dentin surfaces. Upon sound enamel, ordinary fruit acids or vinegar (5 per cent acetic acid) do not produce painful impression. This phenomenon, as expressed in the words of the Prophet: "Every man that eateth of the sour grape his teeth shall be set on edge," may also occur as a somatesthetic manifestation of a disturbed psyche. Treatment: Protection of the enamel defect by mechanical or therapeutic measures.

Reflex Odontalgia.—Genuine neuralgia arising from dental causes is extremely rare, while neuralgiform types of pain in and about the

teeth are more often observed. Neuralgia should be looked upon as a neuropathic disturbance of nerve fibers without having any direct connection with an organic disease. True trifacial neuralgia, sometimes referred to as tic douloureux, Fothergill's disease or prosopalgia, manifests itself by a sudden paroxysmal pain of a sharp, darting, stabbing character, which is most common along the course of the supra- and the infraorbital branches of the left side of the face with increased lacrimation, slight edema, gray eyebrows and convulsive twitches and tenderness at the infra- and supraorbital foramina (points douloureux), as well as along the course of the nerve distribution. It is most often restricted to women of middle age in which neuropathic disturbances or a general disease, principally anemia or hyperesthesia of pregnancy, play predominant roles. Neuralgia should be differentiated from neuritis, *i. e.*, an

FIG. 7.—Typical position of hands in tic douloureux. (Mayrhofer.)

inflammation of a nerve trunk, primarely characterized by continuous pain, impaired sensation, motor paralysis and atrophy. Central trifacial neuralgia, either from involvement of the ganglion itself or its internal roots, or as a result of pressure from a cerebral tumor, *i. e.*, a neuroma, often leads to a faulty diagnosis of toothache. Many patients suffer the loss of one tooth after another in the vain search for the real cause. After the sacrifice of the teeth the dentist or physician may wake up to the fact that the painful disorder is of a central origin, and that a grave mistake has been made.

Neuralgiform types of pain in and about the teeth of an obscure character are occasionally met with. The true cause of the pain may be located anywhere between the origin of the nerve in the brain and its end-organs, *i. e.*, in our case, in the teeth or within their immediate surroundings; however, the sensation of pain is only

manifested at the periphery. If a carefully conducted diagnosis by exclusion has eliminated every one of the above discussed factors, one should be mindful of the possible presence of solid new growths within the body of the pulp, which are referred to as adventitious dentin if attached to the wall of the root canal or as pulp stones or nodules, denticles, internal odontomes, dentinoids, etc., if suspended within its body. The formation of pulp nodules is most likely always due to some type of mild, continuous external irritation which excites the dentin-forming cells of a healthy and, usually, mature pulp to renewed activity. This irritation, however, must never become severe enough to produce acute inflammation. These pulp nodules usually represent pearl-like, shiny beads; they are found singly or multiple and occasionally a few may coalesce into an irregular multiple mass. The pulp chamber as well as the root canals serve as their abodes. Regarding their location in the

FIG. 8.—Pulp stone removed from an upper molar. Normal size.

FIG. 9.—Pulp stone *in situ*. Upper incisor.

various types of teeth, it has been observed that the incisors and canines are relatively seldom invaded, while the pulps of premolars, but primarily the molars in mature age, are principally selected as seats of their formation. Dieck[1] state that he has found pulp nodules in more than 18 per cent of extracted molars and premolars, counting only those teeth which contained macroscopically observable deposits. By their mere presence and close contact with nerve filaments within the pulp these nodules may cause chronic pressure and, as a consequence, more or less severe pain which steadily increases with their growth. Pulp nodules have been observed in root canals which completely occluded their lumen and thereby caused atrophy of the severed pulp stump.

To attempt a correct diagnosis of pulp nodules in a tooth *in situ* is a most hazardous undertaking. The antecedent history furnishes little information; the age of the patient is of some importance, *i. e.*,

[1] Ueber den dentalen Ursprung der Prosopalgie, Wuerzburg, 1897.

pulp stones are rarely observed below twenty. Frequently, the age is the only available clue. Exploration and inspection may bring to light a few additional meager data. The suspected tooth usually shows some external defect which may serve as a focus for the necessary mild irritation which is responsible for the formation of the nodule, *i. e.*, abrasion, erosion or premature senile atrophy of the protecting alveolar process and, occasionally, dental caries. It is characteristic of teeth containing nodules that they present typical senile appearance, and on sectioning always show the zone of translucency. Transillumination is usually negative as the thick body of a molar crown offers a high resistance to the passage of the rays of even a very high power dental lamp. Conductivity of temperature and percussion reveal very little information, although cold applied in the form of the ethyl chlorid spray may cause a painful paroxysm. Dentz[1] lauds the application of cold as possessing specific merits in diagnosing pulp nodules in an otherwise sound tooth. It is claimed that when a tooth responds more painfully on

FIG. 10.—Pulp stones *in situ*. Upper molars.

percussion during a cycle of pain, and this hyperesthesia passes off immediately after the paroxysm ceases, pulp nodules may be suspected. On testing a tooth pulp containing nodules with the electric current usually a slight abnormal sensation is discerned which indicates hyperemia or acute partial pulpitis. However, not too much reliance must be placed upon such slight differences. A good, well-defined roentgenogram is an absolute prerequisite for a diagnosis. The interpretation of a light shadow within a pulp chamber should not, however, be too hastily pronounced a pulp nodule.

The localization of the existing pain and its manifested character is of the utmost importance. In the very early stages of an existing pain about a tooth, one is usually more readily able to distinctly locate the afflicted member. In the later periods there is always a possibility of irradiation. A very carefully conducted inquiry in regard to the subjective symptoms of the existing pain may lead

[1] Tijdschrift voor Tandheelkunde, 1904, p. 231.

to most valuable hints in regard to a differential diagnosis. The patient may describe his pain as being of a severe acute throbbing or lancinating character, often occurring at specific intervals, usually toward evening, or of a milder chronic type. The paroxysm may last for a minute or two, or longer, and the affected side of the face becomes flushed. The absence of convulsive twitches, gray eyebrows, sex, age and the nature of the paroxysm, etc., differentiates dental neuritis from trifacial neuralgia.

To differentiate true central trifacial neuralgia from peripheral dental neuralgiform types of pain, *i. e.*, dental neuritis, local anesthesia is frequently an efficient aid. If during an attack the supposed painful area is blocked out by a local anesthetic and the paroxysm ceases for some hours, one may be reasonably assured that the disturbances lie within the region of the blocked-out teeth, while if the pain presently returns, one has to deal with a central affection of the facial nerve. After the pulp of a tooth has been destroyed, referred pains cease and there are only manifestations of local pains, due to an involvement of the peridental structures.

CHAPTER II.

INFLAMMATION.

Inflammation of the dental pulp in its fundamental principles does not materially differ from the orthodox doctrines accepted at present by the leading pathologists. According to the classic definition of Burdon Sanderson, inflammation may be defined as "the local reaction to an injury," *i. e.*, the reaction of an irritated or damaged tissue. The irritated tissue must, of necessity, still possess its vitality. It is generally conceded at present that inflammation is not, in the strict etymologic sense of the term, a disease; it is the sequence of an irritation which manifests itself in the local defence, *i. e.*, a protective reaction against the invading foe. "The condition of local flaming—*inflammatio*—has, of necessity, been recognized from the very beginning of medical studies, but so long as little was known concerning the causes of disease and less regarding the processes, all that could be accomplished was to regard this as a *state* characterized by certain peculiar symptoms, and the first attempts of a definition, that of Celsus and Galen, so regarded it. Inflammation, they laid down, was a condition characterized by *rubor*, *tumor*, *dolor*, *calor*—redness, swelling, pain and heat—to which definition later writers added a fifth cardinal symptom of *functio læsa*, disturbed function." (Adami.)

Before entering into a detailed discussion of the various manifestations of disturbances of the pulp, it seems advisable to review in a preliminary way the modern pathologic conception of inflammation. Elsewhere the writer has stated that the most important changes occur in the bloodvessels, which are distended by an increased flux of blood; this manifestation is, however, very quickly changed by a retarded afflux. The white corpuscles conglomerate in bunches near the vessel wall, especially in the veins and capillaries, while the red blood corpuscles keep to the center of the blood stream. The leukocytes and the lymphocytes now pass between the endothelial cells through the vessel wall of the veins and of the capillaries, but not of the arteries. This wandering of the white corpuscles—*diapedesis*—is accomplished by a transudation of blood serum, which fills the surrounding tissues, causing an edematous swelling. Later on the red corpuscles follow, but they migrate in much smaller quantities. The nature of the transudate, the quantity of the blood corpuscles and the admixture of foreign bodies determine

the character of the infiltration, as it may be a serous, fibrinous, purulent, hemorrhagic or croupous exudate. Another important, but usually less recognized, symptom of inflammation is the increased osmotic pressure within the infiltrated area. Under normal conditions the osmotic pressure within the soft tissue is promptly regulated by the organism, probably, according to Massart, through specific nerves, *i. e.*, the normal equilibrium of the isotonic index of the blood and tissue fluids remains stationary.

In pathologically altered tissues the composition of these fluids is continually interfered with, and usually results in a marked increase of osmotic pressure, *i. e.*, hyperisotonicity. Increased osmotic

FIG. 11.—Inflamed mesentery of frog: *a*, margination of leukocytes in the dilated capillaries; *b*, migration of leukocytes; *c*, escape of red corpuscles; *d*, accumulation of leukocytes outside the capillaries. (After Ribbert.)

pressure induces pronounced morphologic changes in the cells, and is largely responsible for the resultant pain, followed by inflammation, within the affected area. "We know that the lumina of the vessels in inflamed areas are increased much beyond their normal size, so that the combined area of the lumina of the vessels within the inflamed area is several times the area of the lumina of the vessels entering the part; and the systolic pressure is as much greater in the part as the area of the vessels in the part is greater than the area of the vessels entering it. This is according to a well-known principle of mechanics. To be more definite we may assume the area of the lumina of the entering vessels to be one square foot, and

the area is the same as that on the small area. Therefore, it will be twenty times the smaller pressure (for example, if the smaller is one pound, the larger will be twenty pounds). Thus, it is easy to understand how the increased area of the vessels will indirectly cause the sensation of throbbing. It would further cause compression of any nerve fibers which are found in the organ, and would

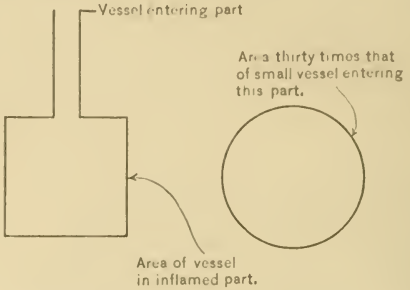


FIG. 12.—Method of pain production in inflammation.

also undoubtedly exert a great pressure upon the capsule. Both of the factors would produce pain." (Behan.) If a simple abscess were taken as an example, the various changes in the tissues could, according to Ritter, be described as follows: In the center of the pus cavity the osmotic pressure may reach a density of 0.6° C. to 1.4° C. (0.56° C. being normal), but in the surrounding hyperemic zone the pressure is less, gradually diminishing in the manifest

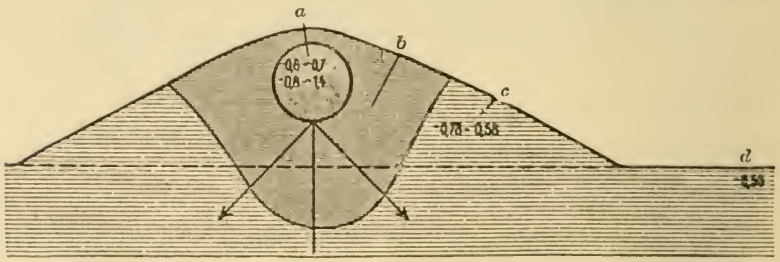


FIG. 13.—Schematic drawing of an abscess. The abscess and the surrounding infiltrated area show the various degrees of osmotic pressure. *a*, abscess; *b*, hyperemic zone; *c*, manifest edema; *d*, latent edema.

edema, and becoming less and less toward the periphery until normal pressure is reached. Aside from the quantitative changes within the inflamed area, qualitative changes of the constituents of the exudate undoubtedly have some important significance. The nature of these latter changes is at present too obscure to allow of any definite statements to be made.

Clinicians recognize two grades of inflammation—the acute and the chronic type. The cardinal symptoms may be readily observed in the acute type, *i. e.*, a quick onset manifesting itself within a few hours or days; while in the chronic type the slow unfolding of the salient features may require weeks and months for their full development with an obscure blending of the various symptoms. Subacute inflammation occupies an intermediate position between these two grades.

Whenever living tissue is injured, whether by mechanical, thermal or chemical means or by parasites, the system at once tries to protect itself against the invading foe by an increased rush of blood into the injured area resulting either in a victorious fight, *i. e.*, complete resolution, or in a surrender to the enemy, *i. e.*, necrosis.

It is generally assumed that the production of disease does not solely depend upon the presence of pathogenic agencies, but that the tissues of the body must be in a lowered state of health which may lead to a predisposition. The principal predisposing factors are hunger, exhaustion, cold and intoxication, although any other possibility, which lessens the natural protecting agencies possessed by the healthy individual or by any of his organs, may create a general or a local lessened resistance.

Local hyperemia, which is the precursor of acute inflammation, results from an increase in the quantity of blood in the injured parts. If it is due to an increase in the flow of the blood it is referred to as arterial or active hyperemia, while, if resulting from an obstruction which retards its outflow it is known as venous or passive hyperemia. In active hyperemia the involved area is bright red in color, and the temperature is slightly elevated and usually accompanied by a marked swelling. Passive hyperemia manifests itself by a bluish-red color (cyanosis) of the involved area, with a somewhat lessened temperature. The veins are distended and an edematous swelling is soon observed, resulting from a transudation of the various constituents of the blood. The cardinal features of the early stages of inflammation, *i. e.*, hyperemia, are the migration of leukocytes, the transudation of serum and the increased activity of fixed tissue cells. At present, it seems proved that the therapeutic benefits derived from hyperemia find an explanation in the bactericidal action of the blood serum. A detailed discussion of the nature of these protective substances—whether they be called alexins, antibodies, agglutinins, lysins, protective ferments, opsonins or phagocytes—would have no direct relationship to our subject at this point. Let it be recollected, however, that Nature utilizes, so far as we know, three important principles of self-protection against local infection: Preparation of the path for the transudation of the serum, positive chemotaxis and increased activity of cell proliferation.

Quite a number of theories have been promulgated to explain the nature of the defensive properties of hyperemia. Buchner claims that the increase of the leukocytes and, in consequence, the alexins are the factors. Hamburger believes that the increased amount of carbon dioxid in the blood, as a sequence of the congestive hyperemia, is responsible and these same views are shared by Chantemesse and Lubarsch. Noetzel also favors this view, provided it is restricted to recent exudates, while Metchnikoff, supported by Leyden, Lazarus and others believes that the phagocytic action of the leukocytes is the predominating factor. It is claimed that the phagocytes break down and thereby liberate active enzymes and ferments which combat the invading organisms and digest the degenerated broken down cells. We cite these theories and leave them without discussion merely emphasizing at this moment the fact that hyperemia is the essential factor which Nature provides in a more or less pronounced degree to combat local infection.

It seems paradoxical to speak of warding off disease by providing inflammation. From a therapeutic point of view, however, inflammation in the past was generally treated by antiphlogistic measures. The layman ripens the abscess with a bread and milk poultice or some similar irritant. From the earliest time heat, in the form of a cataplasm or fomentation, had been applied by the medium of heated rags, stones, china, etc., and has always ruled supreme in the treatment of local infection. Tincture of iodin, the hot-water bottle, the alcohol poultice or the Priessnitz bandage, the therapeutic lamp or the electric-light bath and massage accomplish in reality the same purpose—they produce types of artificial hyperemia. Many of these remedies act only by counter-irritation, producing a secondary inflammation in order to relieve the primary focus of irritation.

Before entering upon a detailed discussion of the inflamatory disturbances of the dental pulp it will not be amiss to reiterate the normal anatomy of this delicate organ as depicted by Professor Hopewell-Smith.[1]

"The dental pulp is a delicate connective tissue consisting of ramified cells imbedded in a slightly fibrous stroma and granular transparent basic substance and is plentifully supplied with blood-vessels and nerves. It is the soft vascular and sentient organ which occupies the central portion of teeth, being naturally bounded on all sides by dentin, which thus constitutes its cavity. There are no traces of any organized lymphatic system in the dental pulp. That is to say, evidence of the existence of endothelially-lined lymphatic capillaries or vessels are wanting. Pericellular and intercellular lymph spaces or tissue spaces are everywhere apparent, as

[1] Hopewell-Smith: The Normal and Pathological Histology of the Mouth, Philadelphia, 1918.

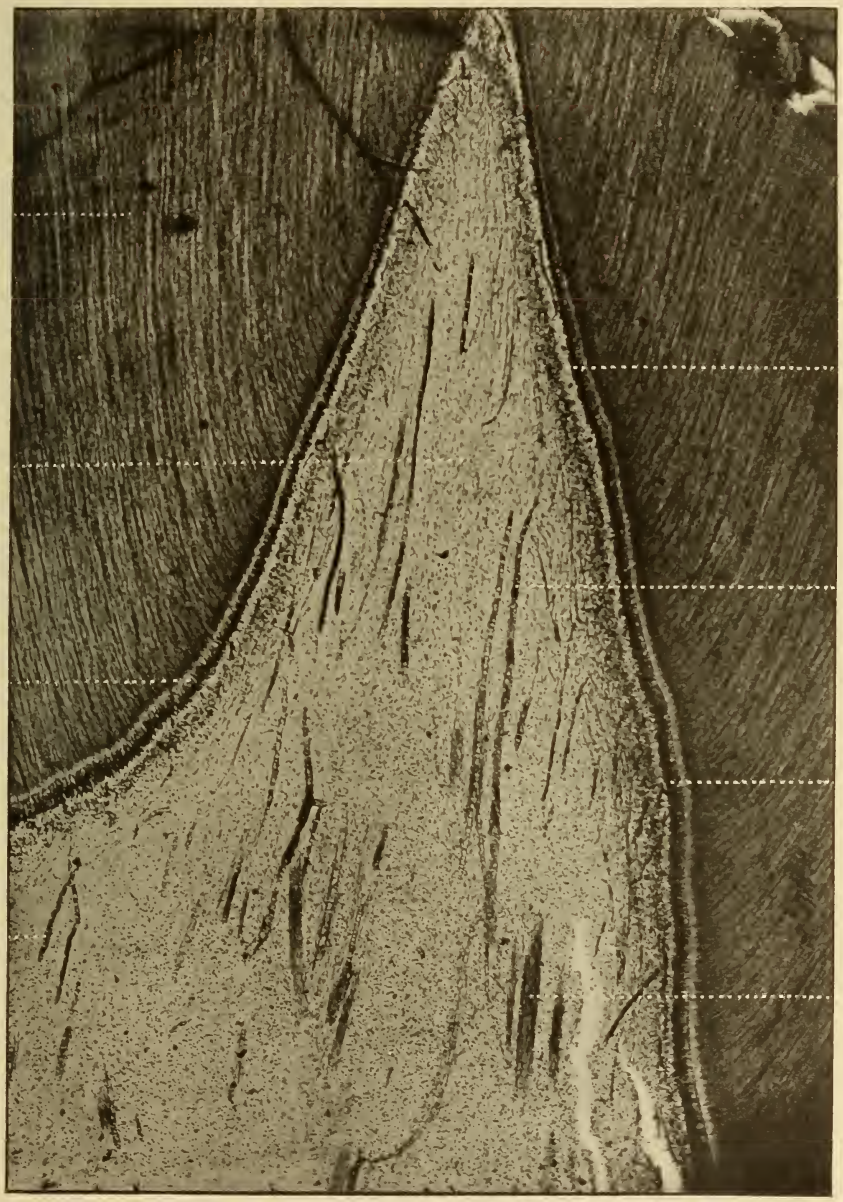


FIG. 14.—Longitudinal section through the cornual region of a young adult molar, the dentogenetic zone of which is on the point of calcification. The pulp is *in situ*. Prepared by Hopewell-Smith's process. Stained with Ehrlich's acid hematoxylene. Magnified 80 times. D. Dentine; P. Pulp tissue in cornu of tooth; D.Z. Dentogenetic zone; O. Odontoblasts; B.L. Basal layer of Weil; B. Bloodvessels; N. Nerve bundles. (Hopewell-Smith.)

also are those around the bloodvessels and nerve bundles. The pulp is saturated with lymph which is derived from the blood plasma, as an exudation from the capillaries. It permeates the pulp tissue and exudes into the dentinal tubules around the odontoblast processes. It, however, does not pass into lymphatic vessels, and does not leave the pulp by such channels. Yet Schweizer, in an elaborate article, claims that by careful injection he has succeeded in demonstrating tufts of lymphatic capillaries in the coronal portion of the pulp, which, collecting the lymph of that neighborhood, convey their contents into one or two wide lymphatic vessels which issue from the apical foramina of the teeth in company with the bloodvessels. The dental pulp is one of those few parts of the body which is devoid of any lymphatic system." (Arthur Hopewell-Smith.)

In a recent publication[1] Kaethe Dewey and Frederick Noyes, of Chicago, have made the assertion that they have been able to verify Schweizer's statement regarding the presence of lymph vessels in the pulp. As yet, their statement has not been generally accepted by dental histologists.

Inflammation of the dental pulp may manifest itself as a destructive or productive process. All acute types of inflammation of the pulp, *i. e.*, hyperemia, simple and suppurative pulpitis, are destructive in their nature, while the chronic types, *i. e.*, ulcerative and hyperplastic pulpitis, represent the productive group. Parenchymatous pulpitis, *i. e.*, the progenitor of the various forms of degeneration, is a non-inflammatory retrogressive metamorphotic process and, as a consequence, cannot be classified under this category. All acute types of pulpitis are extremely labile in their character, *i. e.*, they change very quickly; within a few hours the alteration from an initial obstructive hyperemia into a partial or total simple (serous) or a suppurative pulpitis may take place. The nature of the infection, which in all instances is practically of the same type, *i. e.*, a mixed streptomycosis, apparently plays no part in controlling the final results. A localized abscess may form in one case, usually in a staphylococcus infection, while a widespread phlegmon may occur in another case as a sequence of a streptococcus infection.

The very opposite picture of a destructive, acute pulpitis is portrayed by a productive, chronic pulpitis. The latter disturbance in reality represents *the product* of the inflammatory process and it is very slow in its development, *i. e.*, intensely chronic. The chronic types of inflammation offer excellent opportunities for exhibiting the full power of the defensive properties possessed by the pulp tissue in its struggle for existence. The very young and physiologically hyperemic pulp responds by producing a very vascular new growth of hyperplastic tissue in the open pulp chamber, known

[1] Dental Cosmos, 1917, p. 436.

as pulp polypus, while in the mature tooth this same process of defence usually results in the formation of pulp nodules and adventitious (secondary) dentin.

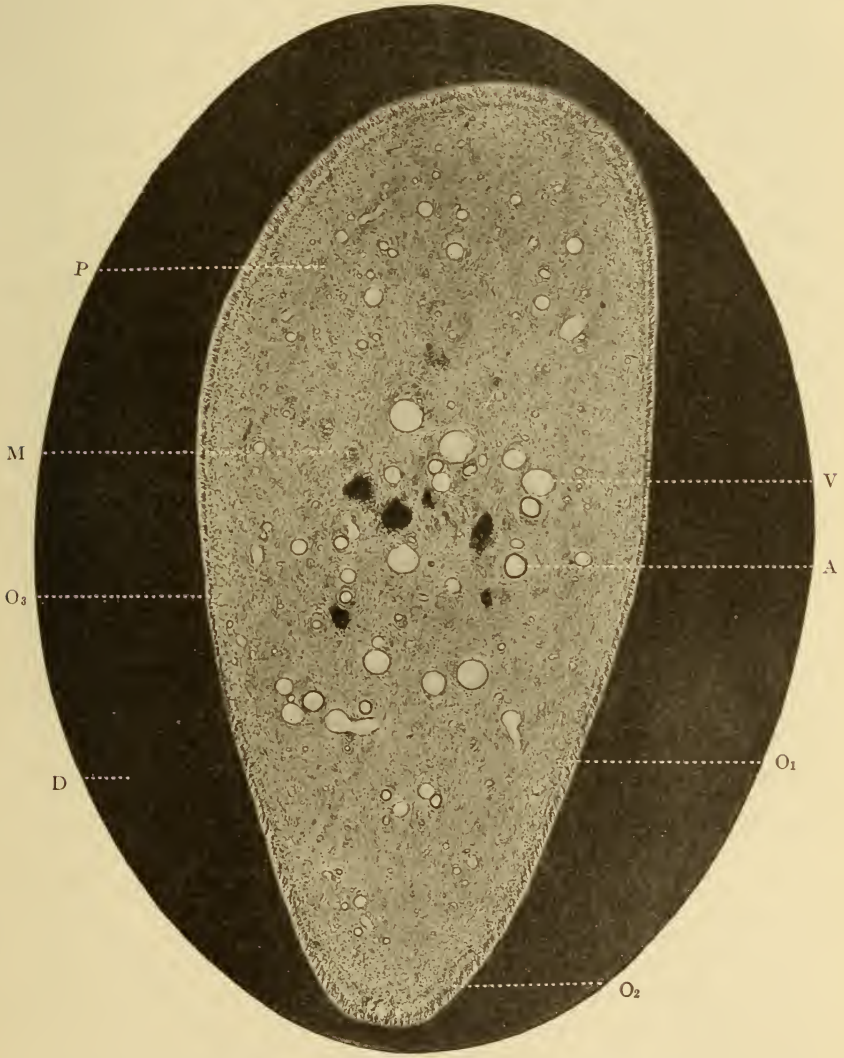


Fig. 15.—Transverse section of an adult canine, with the pulp *in situ*.[1] Prepared by Hopewell-Smith's process. Stained with rubine. Magnified 45 times. D. Dentine; P. Pulp tissue proper; O_1, O_2, O_3. Odontoblast layer; A. Artery; V. Vein; M. Myelinic nerve bundle. (Hopewell-Smith.)

[1] The narrowest diameter of the pulp in the section of which Fig. 15 is a photomicrograph measured 1.5 mm.; the widest diameter 3.35 mm.

Parenchymatous pulpitis, in its very early stages, also assumes the character of a chronic inflammation. Soon, however, the pulp loses all reactive power and from thereon, without even being able to respond to an infection, very slowly degenerates. The degeneration may be a true atrophy, or it may assume a fatty, a fibroid or a calcareous character. Degenerations represents senile metamorphotic changes of necrobiotic tissue.

The anatomic relationship between the dental pulp and its surrounding hard structure, it being tightly enclosed within an unyielding wall of dentin, precludes its physical examination. Therefore, with one exception, we are unable to recognize the classic symptoms of acute inflammation. The one exception refers to the sensorial manifestation of pain as subjectly experienced by the patient. Ordinarily the color of a pulp cannot be observed except in traumatic or pathologic exposures. When the exposure is sufficiently large a bluish-red, pulsating tissue slightly protruding from the cavity may be seen. Whether or not the temperature of an inflamed pulp is higher than normal cannot be adequately ascertained, as our physical apparatus—the thermometer—is too cumbersome an instrument to lend itself with any degree of satisfaction to the caloric inspection of so minute an organ as the dental pulp. Impaired function of the tooth as a sequence of pulpitis does not occur; the tactile sense of the tooth is restricted to its investing membrane, *i. e.*, the pericementum. Consequently, the only symptom left for consideration is the manifested pain. Pain is always present in the genuine types of acute inflammation. It is the result of pressure exerted by the exudate on terminal nerve filaments and additional irritation produced by toxins, acids, enzymes, etc. It proportionally increases with the acceleration of the exudate. The severest type of pain is manifested in suppurative pulpitis. Its character is less severe in chronic types and it is totally absent in true degeneration, as in this condition there is no exudate. Fortunately, the subjective exhibition of pain from an inflamed pulp is so very characteristic in its subtle differentiations that by its guidance we are able to arrive at a fairly accurate differential diagnosis.

If we compare the peculiar anatomic structure of the dental pulp with other soft tissues of the body we are at once forcibly impressed with the enormous difficulties which we are apt to encounter in our humble efforts to apply conservative treatment. Four outstanding negative factors are at once recognizable. As stated (1) the pulp is encased in a hard, unyielding capsule of dentin; (2) no lymphatics are present within its mature structure; (3) it is impossible to establish efficient drainage; and (4) it is difficult to gain ready access to the diseased organ for the purpose of applying remedial measures.

The extravasated serum as it occurs in the early stages of inflammation cannot well be accommodated within its own body, *i. e.*,

the pulp cannot swell and, therefore, the serum quickly spreads over the entire organ. By this increased internal pressure a partial pulpitis very rapidly changes into a total pulpitis, with the increase of the exudate, incidentally containing acids, toxins and ferments, augmented pressure and irritation is exerted upon the nerve filaments which, in consequence, respond with a progressive exacerbation of pain. This phenomenon is peculiar to an inflamed pulp; it is the reverse of what ordinarily takes place in other soft tissues.

A further important factor which markedly lessens the chances of resolution in an inflamed pulp consists, as stated, in a total absence of lymph vessels in the mature organ. Ordinarily, the resorption of the exudate occurs primarily through the lymphatics. If the exudate is not removed the increased internal pressure changes the primary obstructive hyperemia into a more or less complete stasis, *i. e.*, stagnation of nutrition and circulation. As the dental pulp cannot establish a collateral circulation, at least not in the single-rooted teeth, the danger arising from an interference with the nicely balanced blood-pressure of the healthy organ is of enormous significance in regard to its future well-being. A dogmatic biologic law asserts that at no time should the blood current be interrupted; complete persistent stasis always signified death of the involved tissue. By comparing the very small foramina of a fully developed human tooth with the relatively large bulk of its pulp, it is surprising that a resolution of an inflamed pulp ever occurs. If, accidentally, the causative factor of the inflammation of a pulp is of an infectious nature, a collection of pus results which localizes itself into an abscess, but more often on account of the labile nature of the involved tissue it readily spreads over the entire pulp. Aseptic inflammation due to mechanical causes or to chemical substances other than those derived from living organisms may also occur. However, even if primarily no bacteria may have been present, as a rule, sooner or later they make their appearance and the sequences of infection are added to the original insult.

Abscess formation is by no means always the sequence of an inflammatory process. Its termination rests with the degree of the produced inflammatory changes and the quantitative damage involved in the destructive process. The simplest outcome is recovery by resolution. If the disturbance has been of a mild character the exudate is taken up by the lymphocytes and is returned to the circulation while the damaged cells are removed by the phagocytes. Should there be present a preponderance of escaped polynuclear leukocytes the exudate is referred to as pus. The physical nature of the pus is largely dependent on the biologic peculiarities of the different bacteria and their activities upon the surrounding structures. In the ordinary soft tissues the newly

formed abscess terminates the local process, *i. e.*, with its natural rupture or through an incision the confined pus is released, drainage is established and immediate reorganization of the involved area is instituted. In the dental pulp, unfortunately, suitable drainage cannot be established, partially on account of the minuteness of the organ and partially on account of the inaccessibility to the focus of infection. The late W. D. Miller pictured the difficulties of treating an inflamed pulp by citing the following unique comparison. "If we attempted to treat an inflamed finger-tip through a small hole drilled into a thimble placed over this tip we should find it wrought with many difficulties and, most likely, with little hope of success as compared with the treatment of the same condition without the thimble."

Pulp tissue, in many respects, is analogous to bone marrow, and shares with it the very labile nature of this structure. As it possesses only a very low degree of resistance and very little power of reorganization, any pronounced disturbance of its equilibrium is almost certain to cause its death. Even the application of such sedative or antiseptic drugs which, relatively, are usually tolerated by other tissues, have proved to be destructive to this delicate organ.

After the subsidence of the inflammatory process in the ordinary tissues, organization and repair are instituted at once. Granulation tissue is formed, which acts as a strong barrier to absorption and reinfection. Healing of the wound takes place by first or second intention. Both processes are analogous except in the matter of quality formation. Healing by first intention will always occur in a non-infected wound, while in the process of repair by second intention a large amount of granulation tissue is formed. Profuse secretion keeps the walls of the wound clean and active hyperemia of the involved area furnishes the necessary nutriment for the increased cellular activity. Granulations spring from the bottom of the cavity to replace the lost tissue and epithelium, if present, quickly covers its surface. In the injured dental pulp resolution is very rarely observed. Pulp tissue, as stated, consists primarily of embryonic connective tissue, poor in fibers, and it does not contain epithelium. The highly specialized connective tissue of the dental pulp possesses practically no ability of reorganization and, consequently, repair by first or second intention, except in a few isolated instances is practically impossible. In the adult tooth death of its pulp is the usual sequence of an inflammation.

The loftiest aim of applied therapeutics within the province of operative dentistry should find its sublime expression in the conservative treatment of the dental pulp. It is to be regretted that the realization of this ideal, for the present at least, is restricted to very limited bounds. Theoretically, from the viewpoint of experimental surgery, restoration of the function and vital energy of the

diseased pulp should offer no difficulties; clinical experience, however, does not bear out this statement.

Basing his judgment upon the above discussed difficulties as they present themselves clinically and in conformity with the general consensus of opinion of the majority of practitioners, the writer frankly admits that the prognosis of an inflamed pulp is, in most instances, unfavorable. Dogmatically, it may be asserted that a severely inflamed mature pulp is a lost organ and its removal is an imperative necessity for the future welfare of the respective tooth and the individual as a whole. The author does not wish to impress the reader, however, that every case of pulp disturbance *ad hoc* is hopeless and calls for immediate destruction of this organ; he is convinced that certain types of obstructive hyperemia and possibly the very earliest states of acute pulpitis in a young growing tooth are amenable to conservative treatment, and, as a matter of fact, such treated pulps occasionally do respond by resolution. No rule of thumb can be laid down; the judgment of the operator has to decide each individual case in accordance with a carefully made diagnosis and with due consideration of all circumstances. While it is true that the chances of recovery of a disturbed young growing pulp have much in their favor, the same chances in an adult pulp —*cæteris paribus*—are always extremely poor. In the latter cases it may be dogmatically stated that the only absolute safe treatment of a severely inflamed adult pulp is its destruction and removal.

The ruthless destruction of the dental pulp in every given case, as advocated by one group of practitioners, is equally as futile as the extreme conservatism displayed by another group. Here, as in everything else pertaining to the practice of the medical arts, sound clinical reasoning based upon an intimate knowledge of pathologic anatomy should be the guiding post.

The question may be rightly asked at this moment: What value possess the living pulp for the future preservation of the tooth? As an indisputable clinical fact it must be admitted that a pulpless tooth has lost much of its resistance to secondary carious processes. Secondly, the crown of a pulpless tooth containing a necrosed pulp is liable to become discolored and, thirdly, a pulpless tooth, as a point of minor resistance, offers suitable chances for the future development of periapical disturbances. Improved technical methods in the bleaching of discolored teeth have largely eliminated the second objection. The last factor, especially, has, within recent years, given rise to much unsound speculation. Zealous advocates of wholesale extractions have culminated their erratic views by employing such catch phrases as: The pulpless tooth must be eradicated from the human economy, which is equally as hazardous a proposition as the assertation of the extreme conservatives who pretend to save every pulpless tooth. While there are numerous cases of

infected pulpless teeth retained in the mouth which constitute a distinct menace to the general health of the individual, and which as a consequence should invariably be removed, there also exists the indisputable clinical fact that the greatest majority of pulpless teeth by the present improved methods of treatment of infected root canals may be restored to health.

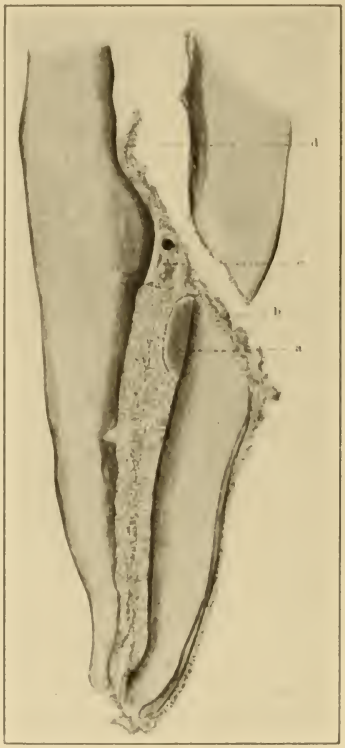


FIG. 16. Exposed pulp protecting itself against infection. *a*, Secondary dentin; *b*, round cell infiltration; *c*, denticle; *d*, chronic pulpitis. (Williger).

Evidence has been furnished by Tomes, Gysi, Williger, Roemer, Roy, Wachtl and others that on very rare occasions a pulp may possess the power of protecting itself against an arising infection. Chronic types of pulpitis, *i. e.*, hypertrophy and ulceration are examples of the gallant struggle of an infected pulp against the invading foe. However, occurrences of this type are extremely rare and attempts of conservative treatment based upon such accidental possibilities are not to be encouraged, except in a few isolated instances in which the general conditions seem to justify the procedure.

CHAPTER III.

ETIOLOGY OF DISEASES OF THE DENTAL PULP.

When a patient suffering with pain in a tooth which contains an aching pulp appeals to the dental practitioner for treatment, it is the paramount duty of the latter to ascertain whether the diseased pulp is amenable to conservative treatment, or whether it should be destroyed. From a clinical point of view it is imperative to ascertain, if possible, the morbific cause of the disease, *i. e.*, its etiology. The early removal of the cause and the prevention of its return as instituted by the physician frequently produces a "cure," while the return of the pathologically altered tissue to normal physiologic function is accomplished by Nature. In the words of Celsus this axiom is expressed as *natura sanat, medicus curat.*

SCHEMATIC OUTLINE OF CAUSES OF PULPITIS.

- Pulpitis
  - Primary causes
    - Causes of a mechanical nature
      - Traumatism, abrasion, calcareous deposits within the pulp, fillings (direct and indirect) and retention pits, correction of irregularities, separation of the teeth, pressure from substances wedged into cavities, etc.
    - Causes of a chemical nature
      - Caustics, protoplasm poisons, products of bacterial metabolism, etc.
    - Causes of a thermal or electric nature.
    - Causes of a parasitic or mixed nature.
  - Secondary causes
    - Causes originating in the pericementum.
    - Causes as a sequence of general diseases.

The causes of diseases of the dental pulp are identical with those which are responsible for the production of pathologic disturbances in other tissues of the body, and, in accordance with the accepted standards of the practice of the medical art, may be referred to as being primary or secondary in their nature. The causes may be mechanical, chemical, thermal, electric or parasitic in their nature. All of these disturbances produce local manifestations. The primary causes, as their name implies, attack the pulp directly, such as an infection from a carious defect; while the secondary

causes select an indirect route, as those arising from a pericementitis or an osteitis.

The vast majority of the causes which are responsible for pathologic disturbances of the dental pulp find their origin in a direct or indirect association with the infective processes of the hard structures of the tooth, *i. e.*, dental caries as a sequence of a streptomycosis of a mixed type. The very same organisms which are closely associated with the carious process are always predominant in an infected dental pulp.

To facilitate the ready classification of the etiologic factors of the disease of the dental pulp from a clinical point of view, the schematic outline on p. 49, as suggested by the late W. D. Miller, will be found to be of service.

Fig. 17.—Typical sport accident. Fracture of upper first incisors. (Mayrhofer.)

Causes of a Mechanical Nature.—Traumatic disturbances of the pulp are manifold in their character and are commonly met with in clinical practice. Such accidents as a fall, a blow, or being hit by some solid object, are of frequent occurrence. Children and those adults who indulge in the various types of sports are prone to be injured about the teeth. On account of their prominence, the upper anterior incisors suffer more often than the other teeth. Sport accidents, such as a fall in skating, etc., usually cause typical fractures of the upper centrals. Accidents, such as the breaking of a tooth during extraction, are also frequent occurrences. During the excavation of a carious defect the pulp is often accidentally exposed. A fracture of the tooth crown may directly expose the pulp, or it may occur in such close proximity to this organ that it becomes advisable to destroy it at once. A careful examination of the broken surface with a magnifying glass and a sterile explorer is of great service in discovering a possible minute opening.

An exposure of the pulp must always be looked upon clinically as an infection, and it should be treated accordingly. If the tooth, as a whole, is loosened, an effort should be made to retain it, if possible, in its normal position by ligatures, splints, etc. After the tooth has again become firm it may be tested by the electric current to ascertain the state of its pulp.

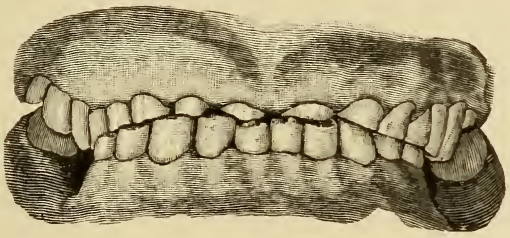


FIG. 18.—Abrasion of anterior teeth, with loss of posterior occlusion. (W. A. Capon.)

Mechanical destruction of tooth structure through abrasion is primarily observed in the aged. In case of loss of the posterior teeth, the anterior teeth are often called upon to bear the entire force of mastication, and as a consequence abrasion of these teeth at their incisal edges takes place. Irritation of the exposed dentin

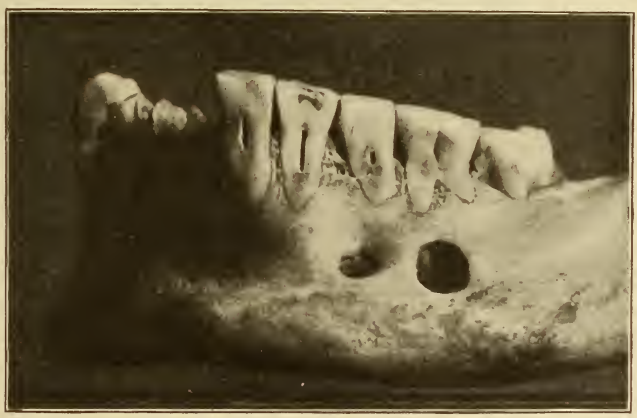


FIG. 19.—Artificial abrasion produced by brushing with a much-used English tooth paste with motor brush for eighteen hours. Remains of gold filling in first bicuspid. (Miller.)

causes a renewed physiologic activity of the pulp which results in the deposition of adventitious dentin in the pulp chamber. Frequently this deposition is insufficient to keep pace with the rapid process of abrasion, and in due time the pulp becomes involved. Certain occupations, such as sand-blowing, glass-cutting, tool-

grinding, etc., in which the atmosphere is charged with abrasive dust, are prone to cause marked wasting of tooth structure. Skulls of Bedouins, as the nomadic tribes inhabiting the sand-saturated atmosphere of the Sahara desert, exhibit pronounced tooth abrasion. The "pipe-stem hole" of the clay pipe smoker and the "blow hole" of the glassblower are typical examples of localized abrasion. Seamstresses, who are in the habit of biting threads, occasionally also exhibit localized abrasion of the first or second incisors, although more often periapical disturbances are observed in these cases from the direct force applied to the long axis of the tooth.

A peculiar type of progressive destruction of tooth structure is known as erosion or, on account of the typical style of cavity produced, as "cuneiform defects." Comparatively little is known of this somewhat common condition. Usually, the typical cavity produced by erosion presents a sharp-edged, smooth, highly polished groove at the necks of the teeth, running transversely to their long axes. Principally the labial and buccal surfaces are involved.

Fig. 20.—Case described as erosion. (Darby.)

In the acute stages irritation of the exposed dentin surfaces is usually painfully noticed by the patient, while in chronic types of erosion a marked absence of pain is to be observed.

Calcareous deposits within the root canal, either as advantitious dentin or in the form of pulp nodules, are of very common occurrence indeed. Occasionally they produce obscure types of idiopathic pulpitis which may baffle the most expert diagnostician.

The irritation produced in preparing a deep-seated cavity for the reception of a filling, and retention pits which are located within close proximity of the pulp, are frequently sources of hyperemia of this vascular organ. In these cases the additional thermal changes of the metallic plug (metals being good conductors of temperature) must be taken into consideration. Intense force employed in malleting gold foil fillings indirectly has caused the death of many pulps.

The too rapid movement of a tooth for the purpose of correcting an irregularity, or the application of quick and intense force in the

mechanical separation of teeth, may occasionally produce severe types of hyperemia or even death of the pulp by strangulation. The use of rubber strips for separating teeth, which was so frequently employed for such purposes in former years, is especially prone to cause pain about a tooth. In removing the rubber dam, small circles may be torn from it and remain under the free margin of the gum, and when left unobserved the resilience of the rubber causes these rubber circles to move upward, impinging upon the peridental membrane, and pericementitis and subsequent pulpitis may be the result. Direct pressure from substances forced into a deep cavity, in which the pulp is exposed or covered by a frail layer of dentin, such as taking an impression with modeling compound, or food crowded into it, may cause pulpitis.

Causes of a Chemical Nature.—Chemical irritants, such as acids, sweets, protoplasm poisons and metabolic products of bacteria are common etiologic factors of pulpitis. It is a well-known clinical observation that a pulp may readily die under a deep-seated cement filling; especially is this accident liable to occur under a silicate filling. Various theories have been promulgated to explain the nature of this accident. The most reasonable explanation is based upon the fact that the cement fluid, which is primarily a concentrated solution of orthophosphoric acid, penetrates through the dentin and in due time kills the pulp. Phosphoric acid does not coagulate albumen, hence its slow penetration by osmosis through dentinal tubules, which are filled with protoplasm in the form of Tomes' fibers, will occur and destroy the underlying pulp by its caustic action. Apparently the respective thickness of the layer of dentin is of little consequence except in the matter of time. In the oxyphosphate cements the action of the acid is largely nullified by its combination with the zinc oxid, while in the silicate cement, in which the fluid is usually present as an acid aluminum phosphate, the acid remains "free" for a prolonged period, and hence its deleterious influence upon the pulp is manifested in due time. Experimental proof of this assertion may be readily demonstrated in the experimental laboratory. By protecting the walls of the prepared cavity with a suitable insulator in the form of a hard varnish, the danger of penetration of the acid is materially lessened.

Sweets, by their hygroscopic nature, are usually only temporary irritants, while certain protoplasm poisons, especially arsenic trioxid may be intentionally applied to destroy the pulp. Impure cement powders containing traces of soluble arsenical compounds are known to have caused death of the pulp. Recent improvements in the manufacture of these cement powders have eliminated the presence of arsenical impurities. If arsenic is present in any one of the various filling materials it will assert its most powerful deleterious effects very promptly. Arsenic present in almost infini-

tesimally small quantities, *i. e.*, 1 part to 1,000,000 parts of the filling compound, is known to have killed the pulp. Acid fumes, especially those of inorganic acids (hydrochloric acid, etc.) are the greatest enemies of tooth structure. They dissolve tooth substance and thereby expose the pulp. Among the organic acids, the damage done by tartaric acid in the so-called "grape-cure" should be mentioned.

Pulp irritation from thermal changes, as stated above, is very common. This is especially true in regard to deep-seated metallic fillings, as metals are excellent conductors of heat and cold. Violent thermal shocks resulting from the injudicious use of refrigerant local anesthetics, *i. e.*, ethyl chlorid, etc., may produce pronounced hyperemia or even strangulation (death) of the pulp with a breaking down of the corpuscular elements of the blood and infiltration of the laked hemoglobin into the tubular structure of the dentin.

Permanent injury and, in many instances, death of the dental pulp resulting from heat evolved during the "setting" of dental cements within a tooth cavity is frequently observed. A few explanatory remarks are necessary for the ready comprehension of this interesting phenomenon. Living protoplasm is killed by heat and the process is *not* reversible. In warm-blooded animals 113° F. (45° C.) is generally considered a temperature at which death occurs in a very few minutes. However, even a temperature of 109° F. (43° C.) may be considered dangerous; of course, it will take a longer time to bring about fatal results. At present, it is generally held that death under these conditions is due to an irreversible heat coagulation of the proteins. The changes in the blood circulation is most pronounced. There is a distinct stasis to be observed in the superficial veins with hemolysis and the first indications of necrosis within the superficial tissue layers. Most probably, poisons are formed as a result of the burn which are particularly toxic to the nervous tissue. The chief cause of death from burns is not to be attributed to the blood changes alone, but theory is in favor of the development of toxic substances. "Death occurs because the vital machinery has been broken down."

During the process of "setting" of certain dental cements rather high temperatures are observed. By employing an automatically regulated thermometer, the writer observed temperature ranges from quantities of freshly mixed cements suitable for an ordinary filling, which registered from 95° F. (35° C.) to 145.5° F. (63° C.) during the period of "setting" lasting from two to five minutes. Cements of the oxyphosphate type are fairly good conductors of heat; if we take the conductivity of gold as a standard, *i. e.*, 100, cements register at about 55 to 60. Dentin ranks about equal with the cements.

From the above discussion it may be assumed that the heat evolved during "setting" of certain cements, combined with the

presence of "free" phosphoric acid, when placed in deep-seated cavities is quite sufficient to permanently injure and, in many instances, kill the dental pulp. The numerous deaths of pulps under cement fillings as observed in clinical practice are sufficient evidence to verify this assumption.

Shocks from electric disturbances within the oral cavity, which manifest themselves in a painful response of the irritated pulp, are quite frequently observed. In cases in which electro-positive and electro-negative metals are simultaneously employed within close proximity as filling materials or as prosthetic appliances, the presence of saliva completes the electric circuit. Gold is an electro-negative metal, while aluminum or amalgam with its predominance of mercury are on the positive side. The interposed saliva acts as an electrolyte, and through ionization a voltaic current is completed which manifests itself in a migration of the positive ions to the negative pole, and thereby a measurable amount of electricity is evolved. Incidentally through the process of ionization of saliva, which always contains sodium chlorid, hydrochloric acid is formed at the positive pole. If, for instance, a gold clasp supporting a denture is placed about a tooth containing a large amalgam filling, the acid set free at the point of contact dissolves the tooth structure, and from the electro-motive force created the gold band may break.

An interesting observation directly bearing upon electric disturbances of the pulp is reported by Prof. Harris.[1] He states that: "In the summer of 1912 I had a lower molar tooth filled with a temporary stopping, consisting of an amalgam of at least three metals, silver, mercury and tin. Within a half an hour of having this inserted I noticed that each time I clenched the jaws at all forcibly there appeared a bright flash of light in the left eye; all through the rest of the day flashes of light, getting fainter and fainter, kept recurring. I noticed that the tooth in the upper jaw which touched the amalgam in the lower was gold-capped. The light experienced was a canary yellow and more like the sensation of a vivid lightning flash (forked lightning) than of any mere luminosity or diffusion of light. So vivid were these subjective flashes that my first thought was that there had been lightning, but on the day in question there was no lightning, thunder or rain. Dr. Frank Woodbury kindly told me that he had known of currents caused in this way being sufficiently intense to give pain in the upper gold-covered tooth when that tooth had an unduly sensitive nerve. I, therefore, suggest that an electric current thus produced was conducted through the bones and tissues of the head and, encountering the retina *en route*, stimulated it to give rise to the

[1] Harris, D. Fraser: Transactions of the Nova Scotian Institute of Science, vol. 14, pp. 47.

subjective sensations of light. The possibility of stimulating the retina *in situ* by electric current was discovered by Ritter in 1800; a constant current passed either transversely across the head in the temporal regions or from the eyelid to the neck will, both at make and break, stimulate the retina causing flashes of light to be perceived. I renewed my acquaintance with these effects by passing the constant current from one dry cell through the eyes transversely across the head; at the make and break the flashes due to this current were less vivid than the flashes due to the tooth current. On using two dry cells, I obtained flashes closely resembling those from the tooth current. I am assured that the current from two dry cells would be painful to the inflamed nerve of a tooth."

Causes of a Parasitic Nature.—Causes of a parasitic nature producing pulpitis are more often met with than any other known disturbance. Bacterial invasion of the dental pulp is probably always due to a mixed infection in which the streptococci are predominating over other organisms. Cognizance must be taken of the chemical metabolic products concomitant with the bacterial invasion, *i. e.*, acids, ptomains, toxins, etc., and the simultaneous action of mechanical and thermal disturbances.

The bacterial invasion of the pulp occurs in the great majority of cases directly from the carious process or indirectly by way of the pericementum near the free margin of the gum tissues. Infectious border-line diseases, which involve the accessory cavities of the nose, and those of the tissues surrounding the teeth, are frequently responsible for the manifestation of secondary pulpitis. Invasion of the pulp by bacteria from hematogenous causes *via* the circulation are comparatively seldom observed.

Secondary Pulpitis, as a sequence of a local or a general disease, is by no means a rare occurrence. In the majority of cases the underlying disturbance is an infection, although general diseases of a non-infectious type, such as leukemia, locomotor ataxia, anemia, chlorosis or increased intradental blood-pressure, etc., may be the exciting factors. Of the infectious diseases, acute nasal catarrh involving the antrum is probably more often concerned with secondary or idiopathic pulpitis than with any other border-line disturbances, although other infections of a general character affecting the mucous lining of the nose or the circulation as a whole, such as influenza, pneumonia, scarlet fever, measles, malaria, etc., must be mentioned. Infectious disturbances of the temporo-maxillary joint, *i. e.*, acute mandibular arthritis, may bring about a secondary pulpitis in the teeth of the lower jaw.

CHAPTER IV.

CLASSIFICATION OF DISEASES OF THE DENTAL PULP.

Concerning a classification of the various diseases of the dental pulp—*nosology*—quite a confusion exists in our current literature, and unfortunately, even in many of our text-books a detailed discussion of this interesting subject is often avoided. Not alone is this apparent laxity of clinical inquiry appalling to the searching student, but he is also confronted with a gross neglect of correct pathologic terminology. Hyperemia, inflammation and gangrene are the usual routine terms selected to designate the bulk of the various types of pulp disorders. Hyperemia has been called irritation of the pulp, which in itself is a misnomer, as irritation is the cause and not the disease. This is equally true in referring to inflammation as a disease. The collective term "pulpitis" is frequently employed to designate generically all states of inflammation of the pulp.

Again, gangrene, a term which is utilized to cover the various phases of dead pulp tissue, is referred to as a disease, while in reality it is the sequence of necrosis, *i. e.*, the necrotic pulp is acted upon by specific organisms, which results in gangrene. Obviously, a dead pulp cannot be "treated," as it is no longer "diseased."

From a purely pathologic point of view, Arkœvy[1] has probably presented, so far as known, the most subtle division of pulp diseases. For clinical purposes, however, this division is too complicated.

[1] Classification of Diseases of the Pulp (Arkœvy).

I. Pulpitis acuta.
   1. Pulpitis acuta septica seu superficialis.
   2. Pulpitis acuta partialis.
   3. Pulpitis acuta totalis.
   4. Pulpitis acuta partialis purulenta.
   5. Pulpitis acuta traumatica.

II. Pulpitis chronica.
   1. Pulpitis chronica parenchymatosa.
   2. Pulpitis chronica totalis purulenta.
   3. A. Pulpitis chronica hypertrophica granulomatosa.
      B. Pulpitis chronica hypertrophica sarcomatosa.
   4. Pulpitis chronica idiopathica seu concrementalis.
   5. Gangræna pulpæ totalis.
   6. Pulpitis chronica idiopathica seu concrementalis.

III. 1. Atrophia pulpæ simplex.
   2. Atrophia pulpæ sclerotica.
   3. Atrophia pulpæ reticularis.
   4. Dissolutio pulpæ absoluta.

Many other classifications have been brought forward, among which we may cite the classic schemes of Rothmann and of Black and those of John Tomes, Wedl, Adolf Witzel, Abbott, Miller, Boedecker, Burchard, Roemer, Walkhoff, Hopewell-Smith, Fischer, Colyer, Kantorowicz and others. To construct a scheme of classification of diseases of the dental pulp which answers all purposes equally well is quite difficult, and is very largely a matter of personal equation. However, there are certain unalterable pathologic facts which must be observed as being fundamentally essential for a suitable division. Ordinarily we refer to a sound, healthy pulp as a normal pulp; this very term, however, needs some further amplification. The tooth pulp should be looked upon as a transitory organ. Normally, with the completion of the tooth its function is accomplished, and from that period retrogressive changes take place within its own tissue which may under suitable conditions lead to complete atrophy or other types of degeneration. At some other period, however, its activity as a constructive organ may be called upon after calcification of the tooth is accomplished. As the result of some pronounced type of stimulation it may deposit adventitious dentin and pulp nodules for the purpose of protecting its own integrity, or some other factors of overstimulation of the exposed pulp may lead to the formation of a hyperplastic growth, *i. e.*, pulp polypus. As it is a very vascular organ, any disturbance of its integrity will manifest itself in the typical reaction of a tissue to an injury—inflammation.

The primary manifestations of the inflammatory process always occur at the place of entry of the disturbance, *i. e.*, usually in the coronal portion of the pulp. As a sequence, a partial acute simple pulpitis is the result. The character of the severity of the infection determines the rapidity of its progress. Within a comparatively short space of time, usually within a few hours, or rarely days, total inflammation of the pulp quickly follows the initial disturbance. To differentiate clinically between a partial and a total acute pulpitis—which, incidentally, is merely a quantitative and *not* a qualitative manifestation of the inflammatory process—is very difficult, if not impossible. From the viewpoint of the clinical pathologist there is no valid reason for such a subtle diagnosis, as the treatment of both grades is virtually the same. The progressive increase of the acute infection leads to the formation of a localized collection of pus, *i. e.*, an abscess, or to a general suppurative infiltration between the spaces of the connective-tissue cells, *i. e.*, a phlegmon. Again a partial, however in most instances a total suppurative, pulpitis is the sequence, which for the above stated reasons need not be diagnosed separately.

Most acute types of pulpitis terminate in a surprisingly short time in necrosis or gangrene. Under certain conditions, however,

the acute form may assume a chronic stage. The prerequisites for the change from an acute into a chronic pulpitis are: a pulp possessed of a high degree of vitality and free drainage of the formed exudate, *i. e.*, an open pulp chamber. The various stages and final results of chronic pulpitis are manifold; usually two types are diagnosed, namely, ulcerative pulpitis and hyperplastic pulpitis. The former type is most predominant in the neglected mouths of children. An intermediate subacute grade of chronic inflammation occasionally occurs, and is known as parenchymatous pulpitis. In most instances the latter is the predecessor of the numerous types of degeneration of the pulp.

As has been stated above, inflammation of the dental pulp may manifest itself as a destructive or a productive process. All acute types of pulpitis are destructive in their nature, while the chronic types represent the productive, often referred to as the "constructive" group. This classification, which is based upon manifested histopathologic symptoms, is still frequently employed by clinicians, and may be depicted as follows:

A. *Acute destructive pulpitis* (the pulp usually is *not* exposed):
   1. Acute simple pulpitis.
   2. Acute suppurative pulpitis.

B. *Chronic productive pulpitis* (the pulp usually *is* exposed):
   1. Chronic ulcerative pulpitis.
   2. Chronic hyperplastic pulpitis.

It will be observed that the above classification does not take any special cognizance of such important clinical manifestations as hypersensitive dentin, the exposed healthy pulp, hyperemia of the pulp, degeneration of the pulp, secondary pulpitis and necrosis and gangrene.

Hypersensitive dentin, when viewed as a clinical entity, may be designated as a state in which the exposed dentin of a vital tooth is painfully responsive to any type of irritation. The physiologic normal dentin has no sensation; by virtue of the innumerable protoplasmic fibers, which traverse it from the pulp cavity to the periphery tactile impressions, thermal changes and chemical or electric irritations are transferred to the anatomic threshhold of sensation, *i. e.*, the nerve filaments distributed at the surface of the pulp and which now react in varying degrees. The response to these various types of irritants is merely an expression of magnified impulses and consequently, in its final analysis, hyperesthesia of dentin denotes an irritated pulp. From the viewpoint of the clinical practitioner, therefore, the treatment of this type of pulp irritation necessitates a detailed discussion.

The exposed healthy pulp in reality should not be included in the discussion of diseases of the dental pulp proper. However, as the exposed pulp, from a clinical point of view, must always be

looked upon as an infected tissue, and one which as a consequence requires treatment, a discussion of the procedure of protecting the exposed pulp is imperative, and therefore its insertion at the beginning of our grouping of the pulp diseases proper seems to be justifiable.

Hyperemia of the dental pulp portrays a clinical manifestation of pulp disturbance which in actual practice is probably more often met with than any other type of disorders of this organ. Its correct diagnosis and the subsequent method of treatment is of the utmost interest to the clinical practitioner.

Clinically, degenerations of the pulp do not form a group of recognizable diseases, as they cannot be diagnosed *in situ*, hence their discussion is of less interest to the practitioner. Generically, they constitute an important part of special pathology, and are referred to in our grouping only from the viewpoint of the general practitioner in order to complete the chain of clinical pathology.

Secondary or idiopathic pulpitis usually manifests itself as hyperemia, or, in rare instances, as any one of the inflammatory types of pulp disorders, or as necrosis. As its name implies, its secondary manifestations are always the sequence of some primary diseases. Clinically, these border-line diseases play a most important role in the routine work of the general practitioner, hence they are entitled to a special consideration.

Death of the pulp, *i. e.*, necrosis and its sequence, gangrene, as stated above, must not be classified as "disease." Clinically, its frequent occurrence and its many sequels are of vital importance to the practitioner, consequently the removal of the dead pulp tissue and the subsequent treatment of the pulpless tooth need to be discussed in detail.

In the following simplified classification it has been our aim to formulate a grouping of the disorders of the dental pulp from the viewpoint of the clinical practitioner. The specific symptomatology of each existing pathologic condition has been selected to serve as the guiding mentor so as to be able to portray the respective ailment as a clinical entity. Its object is to present a working basis for the *ultima ratio* of all clinical procedures, namely, the application of rational therapeutic measures, in the broadest sense of the term, for the purpose of relieving, preventing or favorably modifying the evolution of diseases of the dental pulp.

Classification of Disorders of the Dental Pulp.

I. Types of irritation:
   1. Hypersensitive dentin.
   2. The exposed healthy pulp.
   3. Hyperemia.

II. Types of inflammation (*Destructive pulpitis*):
    1. Acute simple pulpitis.
    2. Acute suppurative pulpitis.
    3. Secondary pulpitis.
III. Sequences of inflammation (*Productive pulpitis*):
    1. Chronic ulcerative pulpitis.
    2. Chronic hyperplastic pulpitis.
    3. Necrosis and gangrene.
IV. Retrogressive metamorphosis of pulp tissue:
    Atrophy and degeneration.

CHAPTER V.

GENERAL PRINCIPLES OF DIAGNOSIS OF DISEASES OF THE DENTAL PULP.

Before entering into a detailed discussion of the clinical application of the diverse methods employed in the examination of a diseased pulp, let us briefly reiterate what is generally understood by "diagnosis." It is the art of distinguishing one disease from another and its object is to enable the practitioner to recognize:

(*a*) The existing disease by a specific name.

(*b*) The respective stage and the peculiarities of the disease.

(*c*) The existing or possible future danger.

From the viewpoint of the dental clinician, it is of interest to ask what practical values are derived from a diagnosis? Some operators make the radical statement, with but slight modification, that a pulp which has been painful for a more or less prolonged period should be devitalized at once without further consideration, and they regard a correct diagnosis of the various diseases of the pulp as being of little practical value. The opponents of such radical views look more favorably on the conservative management of this delicate organ and to them the respective state of health of the pulp is of paramount importance in regard to its further conservative treatment. The ancient medical axiom, "*Qui bene diagnoscit, bene medebitur*," which may be freely translated: "A good diagnostician is a good physician," should also be applicable to the conscientious dental practitioner.

The correct interpretation of the various principles involved in the recognition of disturbances of the pulp are of the utmost importance for the future preservation of the involved teeth. Each definite type of pulp disease requires specific treatment for its amelioration and unless the clinician recognize this fact he does not render his best services to his patients.

The purpose of the practice of clinical dentistry, considering it in its broadest sense as a branch of the healing art, is to institute preventive measures, to relieve suffering and to cure disease. These objects are not achieved by the haphazard utilization of a few stereotyped therapeutic formulas or by certain mechanical procedures, but they are based on a thorough knowledge of clinical pathology.

To support our contention regarding the diagnosis of diseases of the pulp, let us cite a few illustrative cases. Without having made a correct diagnosis, arsenic is placed upon a suppurating pulp. Practically no results are obtained, since this drug is a protoplasm poison which does not act on necrosed tissue. Nevertheless, the drug is made to carry the blame of its failure to act upon the remaining pulp stump. If the necrosed tissue is removed and arsenic is placed on the highly inflamed pulp remnant without having previously relieved the inflammation, intolerable pain is absolutely certain to follow. Again the drug is blamed for its unexpected action. Or a patient may present himself with severe pain in an otherwise sound upper first molar. After a few routine applications of aconite and iodin have been made without obtaining the desired results, it is decided to devitalize the pulp with arsenic. Many visits of the patient are necessary, and the "painless killing" of the pulp turns out to be a barbarous joke, and all this expenditure of time, nerve force and money on account of inexcusable pathologic ignorance on the part of the practitioner is wasted. A diagnostic inquiry might have revealed the fact that this patient suffered from a severe attack of influenza, which is prone to cause hematogenous infection of other tissues, and in this particular case, the pulp of the involved tooth, or teeth, may have been a point of lowered resistance.

A correct diagnosis of a normal, a diseased or a dead pulp *in situ*, is always a matter of great difficulty, and the difficulty is materially increased if the tooth under consideration does not present any visible signs of derangement or if the objective complaints as made by the patient are of an obscure nature.

The intelligent interpretation and the harmonious correlation of the various data as obtained from a verbal examination of the patient and the diverse methods involved in a physical inspection of the diseased tooth tax the diagnostic skill of the operator to the utmost. It should be emphasized that only those who possess a sufficiently broad knowledge of the fundamental sciences of the medical art, an extensive experience and a keen analytic mind, will ever master the difficulties involved in diagnosing the diseases of the pulp in their entirety. The decision of the "lightning" diagnostician should be looked upon with skeptical reserve, and the diagnostic report as rendered *in absentia* by a roentgen-ray laboratory technitian from a roentgenogram with the stereotyped arrow pointing to a spot about the apex of a tooth may be regarded as an aid to, but not as a diagnosis.

Special Procedure in Making a Diagnosis.—In making a diagnosis it is always advisable to follow a systematic scheme in order to avoid mistakes of omission. Suitably printed examination charts in which the obtained data may be quickly entered are valuable

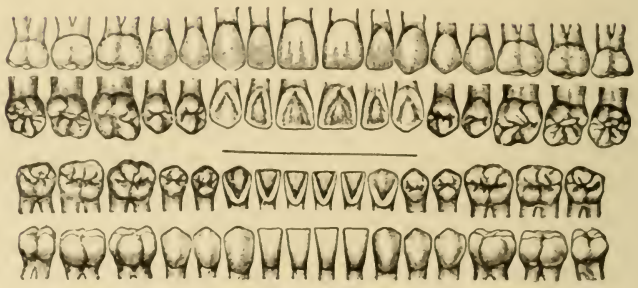
Diagnosis Chart.

Date.....19... No.....

Referred by

Name Address—

Age M.F. S.M.W. Nativity Occupation



Antecedent history
History of present trouble
General health
Mucous membrane
Swellings
Maxillary sinus
Mandibular joint
Condition of the teeth
Character of pain: Lancinating throbbing shooting
dull gnawing spontaneous intermittent
Duration of painful attack Intermission
Pain brought about by: Heat cold pressure
Irradiation: Mental region hyoid region
fronto-nasal region temporo-maxillary region
naso-labial region superior laryngeal region
mandibular region vertical region
Examination of teeth: Exploration color
transillumination conduction of temperature
percussion palpation impaction
occlusion malformation calculus
Faradic test:

R	8 7 6 5 4 3 2 1	1 2 3 4 5 6 7 8	L
	8 7 6 5 4 3 2 1	1 2 3 4 5 6 7 8	

Roentgenogram
Laboratory report: Urine
Blood
Bacteriologic
Pathologic

Diagnosis
Prognosis
Treatment
Remarks

aids in saving time and in preserving uniform records.[1] Aside from the dental diagram and general record columns as ordinarily outlined on these charts, space should be provided for recording the findings of the special examination.

The examination of a diseased pulp should include:

1. The antecedent and present history of the case.
2. Manifestations of pain.
3. Physical examination of the tooth:
   - (*a*) Exploration and inspection.
   - (*b*) Color.
   - (*c*) Transillumination.
   - (*d*) Conductivity of temperature.
   - (*e*) Percussion and palpation.
   - (*f*) Roentgenogram.
   - (*g*) Testing of the pulp by the faradic current.
4. A differential diagnosis.

The Antecedent and Present History of the Case.—The *anamnesis* is of the utmost importance; frequently the decision of a diagnosis hinges upon the facts obtained from an historical examination. Inquiry into possible hereditary taints, such as syphilis (Hutchinson's teeth, necrosis), into the causes of the present disease as a sequence of some previous disease, such as rheumatic affections, colds, antral infection, malaria, influenza, etc. (idiopathic or secondary pulpitis); occupation, as working in acid or match factories (dissolution of the crowns of the teeth, phosphorus necrosis); handling of lead, mercury, bismuth, etc. (stomatitis), glass and sand-blowing (abrasion); glucose and candy-making (candy-makers' caries); turning of mother-of-pearl (pericementitis); clay-pipe smoking (abrasion); chewing of straw and grain (actinomycosis); the use of certain tooth preparations, as charcoal tooth powder (tattooing of the gums); biting of the thread by seamstresses (pericementitis), etc., and specific events in the past or recent history of the patient, including its prodromes and suspected causes, are of diagnostic value.

[1] A convenient way of indicating the various teeth on a record card consists in utilizing a right angle in connection with the number of the respective tooth as employed in the following diagram:

$$\text{R}\ \begin{array}{r|l} 8\,7\,6\,5\,4\,3\,2\,1 & 1\,2\,3\,4\,5\,6\,7\,8 \\ \hline 8\,7\,6\,5\,4\,3\,2\,1 & 1\,2\,3\,4\,5\,6\,7\,8 \end{array}\ \text{L}$$

The horizontal line of the angle indicates whether it is an upper or a lower tooth, while the position of the perpendicular line designates the right or left side of the jaw, *i. e.*, $\underline{6|}$—upper right first molar; $|\overline{3}$—lower left canine, etc. Roman figures may be used to indicate temporary teeth and the mathematical sign $\surd$ may be employed to designate roots. Or the first capital letter of each tooth may be used by placing the number of its position in the arches at its respective corner. Thus the upper right first molar is designated as M^1; the lower left second premolar as P_2, etc. For the designation of the temporary teeth, Roman figures may be employed, *i. e.*, the upper right second incisor, J^{II}.

Manifestation of Pain as subjectively expressed by the patient provides the diagnostician with the most reliable information concerning the nature of the existing ailment. The importance of its interpretation must be obvious as almost 90 per cent of all diseases either begin with, or have, pain as a prominent symptom at some time during their course. Pain is an expression of disease; it is the conscious manifestation of a morbid change causing distress, or when extreme, agony, within the sensory nerve centers brought about by some form of irritation, and it is always referred to the periphery. It is a sensation feared by man, the alleviation of which is constantly attempted by the physician, although it is his most reliable assistant. It is the monitor of approaching ill, and, incidentally, it is the criterion of the intensity of a traumatic insult. To its relentless demands the most obstinate patient has to submit. Unconsciously, the patient seeks to protect a painful organ.

"Pain is a severe but necessary law of Nature, but, like all her laws, it is undeviating in its course; it appears not only as a beneficient monitor, but also as a useless tormentor. Pain is necessary for guarding us in the fight against the forces of disease; it precedes or accompanies the outbreak of most diseases, and warns us that the body is sick and needs attention, although often it may be absent in the most dangerous diseases, thus giving the patient false assurance. The outward expression of pain is by no means a guide to its actual intensity, as pain largely depends upon the psychic condition of the patient." (Goldscheider.)

An analysis of pain should be begun by differentiating its properties, *i. e.*, quality, intensity and duration and by having the patient point out exactly the spot or the region in which it is felt. If the pain is of a radiating character it is necessary to differentiate between the painful focus and its peripheral radiations. Frequently it will be observed that the painful focus coincides with the area in which the pain was localized at the beginning of the attack. However, occasionally a pain may radiate in various directions and may run a most irregular course. Incidentally, with the location of pain, the time of its appearance deserves some consideration. The onset of pain may be associated with some definite hour of the day, or it may be coincident with and dependent on certain occurrences, such as the ingestion of food, the application of heat or cold, etc. The duration of the painful sensation must also receive due attention.

"The intensity of pain, judged from its quantitative variations, depends partially on the intensity of the irritation causing it and partially on the psychic characteristics of the patient. The same etiologic stimulus, which may be endurable to one person, may seriously disturb the psychic equilibrium of another. This double dependence of the intensity of the painful sensation on stimulus and irritability, and the impossibility of projecting externally the

psycho-chemic events in the sensory nerve substance that take place when pain is experienced, render illusory an attempt to estimate the quantity of the sensation for diagnostic purposes." (Schmidt.)

The quality of the pain as expressed by the patient is a relative term, although frequently much information may be gained from the description which he gives. Such descriptive terms as pain of a boring, throbbing, fulgurating, lancinating, radiating or diffuse character, or many other similies, such as gnawing, jumping, etc., are merely clinical expressions of the quality of the painful sensation. An exact analysis of the existing pain demands an accurate determination of all the factors which influence the intensity of the sensation, either in the positive or the negative. Such modifying factors are intimately connected with the causative condition, and are, therefore, of the greatest importance from the viewpoint of the diagnostician. Various modifying agencies greatly influence differences in the individual painful susceptibility of certain parts or of the body as a whole. Psychic disturbances, *i. e.*, excitement, suggestion, diversion of attention, race, sex, age, education, self-control, etc.; mechanical, *i. e.*, position of the body, motion, percussion, pressure, etc.; thermal, *i. e.*, changes of the weather, cold, draft, etc., and chemical, either dietetic or remedial, are important controlling factors of psychic evolution. There may also be associated manifestations of a subjective or objective nature. While some of these manifestations may be remote in their nature, as, for instance, vomiting in severe neuralgia, others may be interpreted as being actual symptoms.

In regard to the kind of pain as subjectively expressed by the patient, it is well to interrogate him by a set of routine questions, as, for example: At what side of the jaw is the pain felt? Which is the painful tooth? When did it begin to ache the last time? How long did the pain last? Does cold or hot water hurt? Does the tooth ache on closing the jaws? Does it feel longer than the other teeth? Does the pain increase on lying down or in coming from the outside cold into a warm room? If a tooth is filled, additional inquiries are necessary, *i. e.*, Had the tooth ached before the filling was placed? Did it pain during filling or soon thereafter? Was the "nerve" destroyed? If a carious defect is present in the tooth under consideration, the application of certain chemicals to the carious spot also may furnish fairly valuable diagnostic hints. The application of a few drops of a 5 per cent formaldehyd solution or of diluted alcohol (50 per cent) into a deep-seated cavity will call forth a series of painful reactions. Depending on specific conditions, as, the presence of acute febrile diseases, especially malaria, influenza and rheumatic affections, of neuralgic tendencies, anemia, menstruation, pregnancy, etc., other questions, of course, will suggest themselves.

An important contribution to the comprehension of the compli-

cated subject of reflex odontalgia, from a clinical point of view was made by the English neurologist, Henry Head, some twenty-five years ago.[1] By a carefully conducted series of observations extending over many years, this experienced clinician was able to show that there existed a definite relationship between certain visceral diseases of the head, throat and abdomen with well-marked zones or areas of superficial tenderness on the skin in the neighborhood of the affected organs. Head postulates, that segments of the spinal cord, which receive painful impressions from internal organs along their sensory track, become painfully altered. Any other sensory impulse, which is conducted from the surface of the skin to the same segment must, necessarily, increase the existing alteration. The

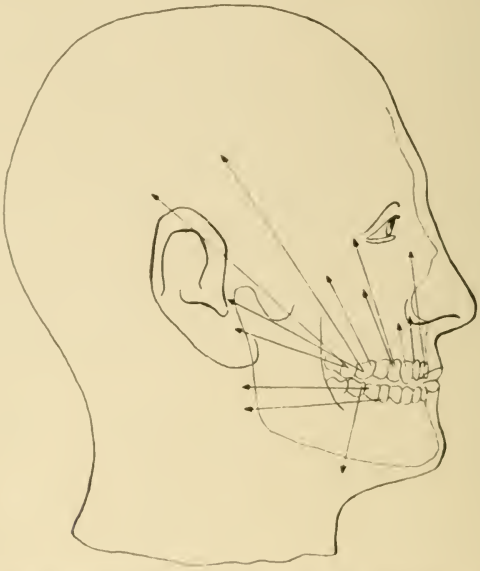


FIG. 21.—Head's reference areas as related to the teeth. (Feiler.)

resultant increased sensorial impression is conveyed to the cerebrum and is recognized as a proportionally increased painful sensation. Diseased teeth bear a definite relationship with one or more surface areas. Head assumes that "To these areas, pain is referred, and over them the skin may become tender when the normal condition of that organ is disturbed." Head's teachings of the location of painful areas about the face as related to the teeth is not yet universally accepted; nevertheless, it constitutes one of the most important contributions to neurology and it is of inestimable value in the diagnosis of obscure reflex odontalgias.

[1] Head: Distribution of sensation with special reference to the pain of Visceral Diseases. Brain, III., 1894.

Head has divided the general topography of the head and neck in regard to referred painful conditions as associated with the teeth into reference areas. According to his conception, each tooth seems to have a separate area of pain reference, *i. e.*:

Upper Jaw.

Tooth	Reference area
1. Incisors	Frontal region.
2. Canine	Naso-labial region.
3. First premolar	Naso-labial region.
4. Second premolar	Temporal or maxillary region.
5. First molar	Maxillary region.
6. Second molar	Mandibular region.
7. Third molar	Mandibular region.

Lower Jaw.

8. Incisors	Mental region.
9. Canine	Mental region.
10. First premolar	Mental region.
11. Second premolar	Hyoid or mental region.
12. First molar	Hyoid region; also in ear and just behind angle of the jaw. The tip of tongue on the same side is also tender.
13. Second molar	
14. Third molar	Superior laryngeal area.

The general topography of the principal clinical painful areas is as follows:

I. **Fronto-nasal Area.**—The maxillary incisors refer their sensation to this area, being situated about $\frac{3}{4}$ inch outward from the median line of the face and extending about $2\frac{1}{2}$ inches above the root of the nose. Its maximal point may be found upon the orbital ridge of the frontal bone about $\frac{1}{2}$ inch from the median line.

II. **Naso-labial Area.**—The maxillary canine and first premolar refer their sensation to this area, covering a part of the upper lip, the lower lip and the under surface of the nose and a part of the cheek. Its maximal point may be located upon a horizontal line extending about $\frac{1}{2}$ inch from the corner of the mouth.

III. **Temporal and Maxillary Areas.**—The upper second premolar refers its sensation to the temporal and sometimes to the maxillary area, however in no case is there a simultaneously existing hyperalgesia in both areas. The first molar is claimed to refer its sensation to the maxillary area only. The maximal point of the temporal area is to be found in the temporal fossa, immediately above the upper border of the zygoma. The maximal point of the maxillary area may be located a little below and to the left of the infra-orbital foramen.

IV. **Mandibular Area.**—The upper second, and in most instances, the third molar, refer their sensation to this area, although the third molar occasionally reflects its sensation to the hyoid area.

The maximum point of the mandibular area is to be found on a vertical line from ½ to 1 inch in front of the tragus of the ear.

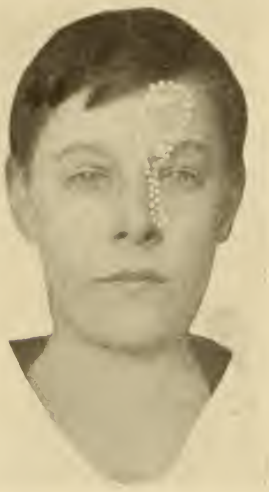


Fig. 22.—The fronto-nasal area (Maxillary incisors.) In this and the succeeding figures the "maximum spots" of intensity of pain are indicated by a round white dot.

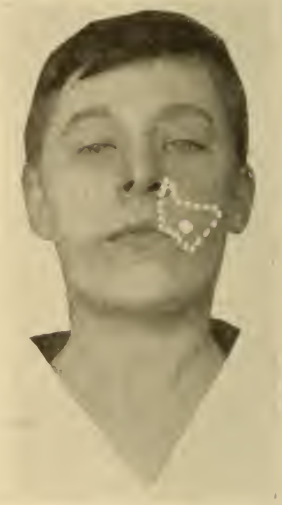


Fig. 23.—The naso-labial area. (Maxillary canine and first premolar.)

Fig. 24.—The temporal area. (Maxillary second premolar.)

Fig. 25.—The maxillary area. (Maxillary second premolar or first molar.)

V. **Mental Area.**—The two mandibular incisors, the canine and the first premolar refer their sensation to this area which covers a rough triangle, including the mental foramen. Its maximal

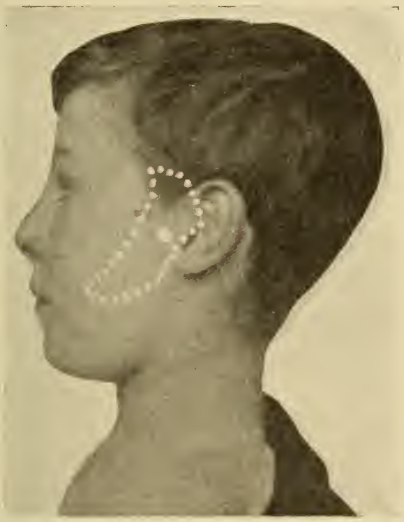


FIG. 26.—The mandibular area. (Maxillary second and third molars.)

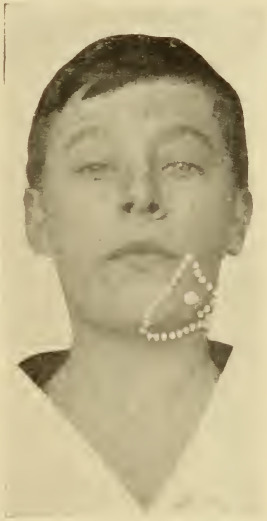


FIG. 27.—The mental area. (Mandibular incisors, canine and first premolar.)

FIG. 28.—The hyoid area. (Mandibular second premolar, and first and second molars.)

FIG. 29.—The superior laryngeal area. (Mandibular third molar.)

point is situated on a vertical line dropped from the angle of the mouth and slightly in front of the mental foramen.

VI. **Hyoid Area.**—The second premolar usually refers its sensation to the hyoid area, although occasionally it may be referred to the mental area. It covers a part of the ramus and it partially reaches behind its posterior border, extending up to the mastoid process of the temporal bone. Maximal painful points may be located in the external auditory meatus, frequently giving rise to earache and at other times directly below the angle of the mandible.

The first and second lower molars also refer their sensation to the hyoid area.

VII. **Superior Laryngeal Area.**—The third lower molar refers its sensation to this area which practically covers the sternocleidomastoid muscle and its maximal point may be located directly in front of the anterior border of the above muscle, *i. e.*, near the laryngeal protuberance. Occasionally reflex pain arising from a lower third molar may be referred to a rather distant zone, *i. e.*, the *vertical area* located at the top of the skull either at the right or the left from the median line and on a connecting line drawn from the upper tips of the ears.

Absence of pain, or analgesia, in otherwise normally sensitive structures, should also be noted. "Analgesia may be either central or peripheral. When central, the lesion may be in the brain or spinal cord. When it is in the brain it may be either endogenous or exogenous. Endogenous analgesia is present during severe emotion, such as great joy, anger and fear, as is seen in the disappearance of a toothache as soon as the patient enters the dentist's office, or the cessation of pain when the patient is in mortal terror. It may be present during arduous mental work which requires great concentration of thought, and also in states of mental exaltation, such as exhibited by religious zealots, examples of which are the Buddhist fakirs." (Behan.) Complete analgesia of the dental pulp may be a most early important symptom of locomotor ataxia.

Exploration and Inspection.—A careful examination of the teeth by exploration and inspection cannot be overestimated as failure to discover even a small defect or lesion might result in subsequent serious consequences. Exploring instruments of various angles and curves are available for such purposes. Mouth mirrors, either plain or with a concave surface, a small magnifying glass, silk floss to detect lesions or roughness on the approximal surfaces of the teeth, an air syringe and absorbent paper to remove moisture, napkins, or the rubber dam to keep the parts dry, etc., are all necessary adjuncts in conducting a careful examination. Preferably, the examination is started at the median line of the left upper arch, examining each tooth in succession back to the last molar and then beginning again at the median line of the right side in the same

manner. The lower arch should then be examined similarly. All the defects of each individual tooth should be marked at once on a suitable chart.

Color.—The color of a tooth varies within very narrow limits. A differentiation should be made between superficial stains as caused by tobacco, chemicals, food, drugs, etc., and bodily discoloration, as a sequence from death of the pulp. Superficial discoloration may be brought about directly by the various filling materials or indirectly by the formation of metallic salts and oxids from amalgams, metallic posts, etc., by recurrent caries under the fillings or by leakage of the filling. Death of the pulp usually produces various shades of darkening of the tooth, ranging from a brownish-yellow discoloration to a slaty-gray or almost black color.

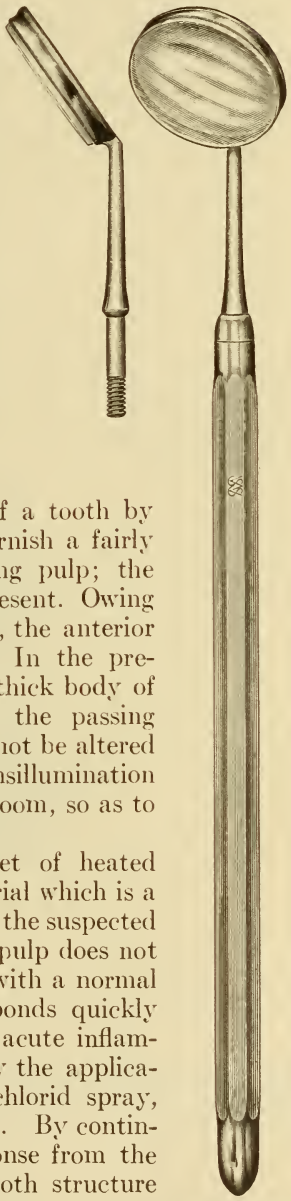


Fig. 30.—Magnifying examination glass.

Transillumination.—Transillumination of a tooth by means of an electric mouth lamp may furnish a fairly well-outlined shadow picture of its living pulp; the picture is diffused when a dead pulp is present. Owing to the variety in thickness of the crowns, the anterior teeth are more easily transilluminated. In the premolars and especially in the molars the thick body of the tooth crowns materially obstructs the passing rays. The transparency of a tooth may not be altered perceptibly by the death of its pulp. Transillumination should always be conducted in a dark room, so as to intensify the shadow picture of the pulp.

Conductivity of Temperature.—A pellet of heated gutta-percha, or some other heated material which is a poor conductor, is placed on the surface of the suspected tooth. A tooth with a diseased or dead pulp does not respond in the same manner as a tooth with a normal pulp. A suppurating pulp usually responds quickly to a temperature above the normal; an acute inflammation of the pulp is often recognized by the application of cold water or the ether or ethyl chlorid spray, while a dead pulp does not respond at all. By continuity the heat rays may produce a response from the pericementum. The thickness of the tooth structure and the presence of various filling materials may, according to their physical nature, increase or decrease the conductivity of temperature changes.

Walkhoff makes the statement that the normal pulp will not react between 50° F. (10° C.) and 120° F. (49° C.). Pain produced

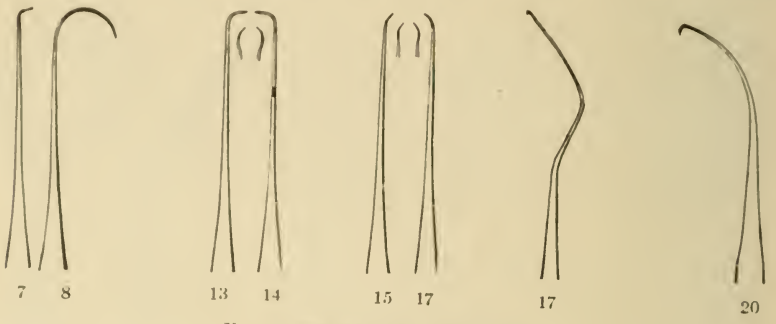


FIG. 31.—Exploring instruments.

by water below 90° F. (32° C.) indicates, according to his conception, inflammation, while pain produced by water heated above this temperature indicates a suppurative condition. A most

FIG. 32.—Cameron's opalite lamp.

interesting exhibition of the thermal reactions of the pulp has been given by the late Louis Jack. He has shown that some individuals readily tolerate temperature changes as applied to their

FIG. 33.—Cameron's diagnostolite.

teeth between 32° F. (0° C.) to 75° F. (24° C.) below and between 118° F. (48° C.) to 135° F. (57° C.) above the normal temperature

of the body. Some patients will experience pain between 75° to 77° F. (24° to 25° C.), while others will not complain even above 145° F. (63° C.).

Percussion and Palpation.—Percussion is best performed by striking the tooth a sharp blow with the butt end of a steel instrument. The peculiar dulness of the resultant sound of a tooth with a suppurating or dead pulp as compared with that of a normal tooth can be usually distinctly discerned by the trained ear. The dulness of the sound is brought about by inflammatory changes of the peridental membrane as a sequence of a diseased or dead pulp or from external causes. The infiltration of fluid between and in the fibers of this membrane changes the relationship of the tooth to the alveolar bone, and consequently the sound waves produced by the tapping have not that full, clear tone which we perceive from a similar percussion of a tooth with a normal pulp, or one that is merely diseased without having affected the peridental membrane. If the finger ball is placed over the apical region of a tooth afflicted with a marked absorption area of its alveolus and its crown is tapped with a steel instrument, a characteristic thrill, known as fremitus, is imparted to the finger-tip, which is absent in a tooth encased in a healthy bony structure.

Roentgenograms.—Roentgenograms are always serviceable, and should be insisted upon in every case of doubtful diagnosis. The correct interpretation of a roentgen-ray picture comprises a thorough familiarity with the normal anatomy and its variations and with the clinical pathology of the involved structure combined with a comprehensive knowledge of the appearance of these same structures under the roentgen rays. It must be clearly understood that a roentgenogram merely depicts a verified gradation of shadow casts produced by the passage of the rays through substances of different density. Dark areas on the negative indicate places lessened in their density, *i. e.*, the rays pass through these rarefied tissues more readily than through the surrounding structures. The roentgenogram does not indicate the physical nature of these rarefied areas.

Ordinary roentgen films or plates do not offer any perspective view of the object, as only two dimensions are portrayed in the picture. For routine diagnostic purposes such views answer quite well. A more accurate knowledge is gained from stereoscopic pictures or from two or more roentgenograms taken at different angles.

To facilitate the intensifying of the very important minute details which naturally will assist in the correct interpretation of the roentgenogram, numerous devices have been suggested. The principle involved in the use of these apparatuses centers about the advantages obtained from the intensified contrasts between light and darkness and in the magnification of the object. The Columbia

Magnifying Radioscope and the Blum Binocular Dentascope are useful instruments for such purposes.

Acute types of periapical disturbances of very recent date are usually not discernable in the roentgen-ray picture, since rarefaction of the involved areas as yet has not occurred. Chronic dis-

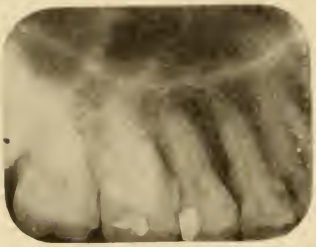


Fig. 34.—Normal bone tissue about the upper molars.

Fig. 35.—Normal bone tissue about the lower molars.

Fig. 36.—True gemination of upper first incisors.

Fig. 37.—Fused upper first incisor.

Fig. 38.—Palatal root of upper molar overlapped by distal root wrongly diagnosed as granuloma.

Fig. 39.—Chronic abscesses.

turbances of the tissues surrounding the root of a tooth are more or less pronouncedly manifested since the existing osteitis offers less resistance to the passing rays. The disturbances include chronic proliferating pericementitis, either singly or associated with osteitis, with a granuloma or with a cyst. The suppurative type

with a rarefied area about the apex of a tooth which is filled with pus, or pus discharging through a fistula, is clearly depicted. Pyorrhea alveolaris always shows marked areas about the affected teeth

FIG. 40.—Chronic abscesses and granulomas.

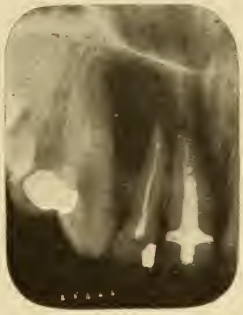


FIG. 41.—Radicular cysts.

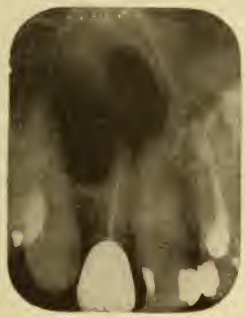


FIG. 42.—Radicular cysts.

FIG. 43.—Pyorrhea alveolaris.

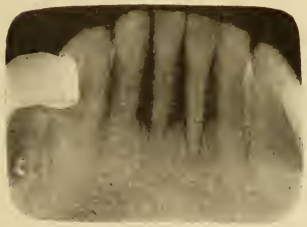


FIG. 44.—Pyorrhea alveolaris.

FIG. 45.—Split root.

and the involved bony structure; this is equally true of a granuloma, a cyst and premature or senile alveolar atrophy.

Electric Examination.—A weak faradic current, as induced by the induction coil in its passage through the body of a vital tooth,

produces a more or less pronounced reaction which is an expression of the sensorial innervation of its pulp. By carefully gaging the current, the resulting irritation expressed as sensation becomes the most valuable diagnostic agent in determining the stage of the vitality of the pulp. The information obtained with the electric pulp tester is so very gratifying that the method of its application needs to be discussed in detail.

Fig. 46.—Columbia magnifying radioscope.

Fig. 47.—Blum binocular dentascope.

Electric Pulp Tester and Its Accessories. — *History.*—In a remarkable book, *Treatise on Dental Caries*, by Magitot (Paris, 1867) and translated by Chandler (Boston, 1878), the following statement is recorded: "This examination (of the dental system), under circumstances so obscure, demands careful attention . . .

Another method has also been proposed; it consists of causing an electric current to pass along the whole extent of the dental arches by means of one of the little induction apparatus so frequently employed nowadays in medicine. By the passage of a current so feeble as not to cause of itself any pain, the carious tooth will become the seat of an acute and clearly localized pain."

John S. Marshall, in a paper entitled "Electricity as a Therapeutic Agent in the Treatment of Hyperemia and Congestion of the Pulp and the Peridental Membrane," makes the following statement: "As a means of diagnosis in obscure cases of the vitality or non-vitality of the dental pulp, I know of nothing so sure to demonstrate to a positive certainty these conditions as the electrical currents, both the galvanic and the faradic. In the more obscure cases, however, the faradic is superior to the galvanic, for if there is the slightest vitality remaining in the pulp it will demonstrate it instantly by causing a response in the tooth."

In 1896 Woodward[1] demonstrated the following: "If a few cells of a cataphoric apparatus are in action, and the positive electrode be applied to the dentin or metallic filling in a vital tooth, while the negative pole is at the cheek or wrist of the patient, a distinct sensation should be felt, while in case of a dead pulp there will be no response; usually even a small filling will transmit a distinct shock in a vital tooth, which is absent in a devitalized tooth. A mild interrupted current has also been used for the test."

The recommendation of testing the pulp by the electric current has never received the indorsement of the dental profession which it justly deserves. In 1902 Fuyt[2] published his researches: "Concerning the use of weak interrupted currents for the purpose of locating certain diseases in the pulp." About the same time, but independent of Fuyt, Hafner[3] utilized the reduced direct current for this same purpose. A year before the publication of Fuyt's and Hafner's observations, Schröder had used the secondary electric current for diagnosing diseases of the tooth pulp and he published his observations in the annual report of his institution. Since then quite an extensive literature on this interesting subject has appeared, the most important publications being those of Witthaus,[4] Grevers,[5] Hamburger,[6] Frohmann,[7] Hesse,[8] Ander Lahn,[9] Schröder,[10] Tousey,[11] etc.

[1] Woodward: Proc. Philadelphia Academy of Stomatology, 1896.
[2] Fuyt: Zahnärztliche Rundschau, 1902, p. 533.
[3] Hafner: Schweiz. Vierteljahresschrift für Zahnheilkunde, 1902, No. 4.
[4] Witthaus: Deutsche Monatsschrift für Zahnheilkunde, 1907, No. 11.
[5] Grevers: Dental Cosmos, 1903, p. 58.
[6] Hamberger: Deutsche Monatsschrift für Zahnheilkunde, 1907, No. 6.
[7] Frohmann: Ibid., 1907, No. 3.
[8] Hesse: Ibid.
[9] Ander Lakn: Oesterr.-ungar. Vierteljahresschrift für Zahnheilkunde, 1907, No. 2.
[10] Schröder: Der Inductionstrom als Diagnosticum in der Zahnärztlichen Praxis, 1907.
[11] Tousey: Dental Cosmos, 1909, p. 513.

It is interesting to note that the various observers differ as far as the nature of the electric current is concerned. Fuyt advises the primary current and Schröder uses the secondary current of the faradic battery, while Hafner advocates the reduced direct current. The alternating current cannot be used for such a purpose unless a generator be interposed to change it to a direct current. All investigators, however, obtained precisely the same results. To judge from the various publications on the subject, coupled with our observations, the primary and secondary combined faradic current is best suited for this work on account of the simplicity of the apparatus and the easy manner in which this current can be regulated.

Fig. 49.—Induction coil pulp tester.

The Faradic Current and Its Accessories.—The faradic battery delivers an easily controlled current of minute quantity. Two forms of induction coils, in connection with the battery, are in general use—the induction coil with a core shield (the tube of Ducheme) and the sledge induction coil of Du Bois-Raymond. The source of electricity for the smaller induction coil is usually received from a single dip battery (acid potassium bichromate solution) or an ordinary dry cell, while the sledge induction coil may be fed from a series of batteries, or from the street current reduced by a rheostat. The small induction coil with one dry cell battery gives universal satisfaction and on account of its cheapness, simplicity and easy transportation deserves to be recommended. More complicated types of apparatuses may be obtained from electric or dental supply houses. The induction coil produces a secondary current in a circuit placed near to, but not in contact with, the galvanic field. This galvanic field, the primary current, is represented by three or four layers of coarse wire wound about a hollow non-conducting cylinder, and the two ends of which are united

with the binding posts. Within the cylinder is found a core of soft iron rods, which are covered in the simple induction coil by a movable brass tube (the tube of Duchenne). Outside of the core and the primary current is a second coil, usually consisting of a

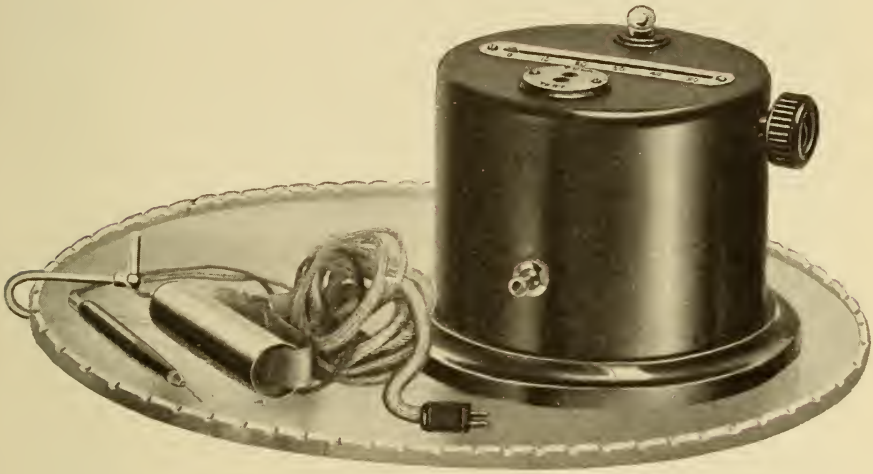


FIG. 49.—Columbia portable electric pulp tester.

FIG. 50.—Columbia pulp tester and ionization apparatus. (Wall model.)

great many turns of fine copper wire. The ends of this coil are also connected with the binding posts. When the current from the cell passes through the coil of coarse wire—the primary current—a current is also induced in the secondary coil of fine wire because the passage of the primary current makes the iron core strongly

magnetic. A vibrator is placed in close proximity to the iron core. When the current passes through the primary coil and becomes magnetized, the steel spring of the vibrator is attracted and breaks the current. The magnet is now immediately released and the spring reasserts itself. The control of the current is guided by moving the brass tube; the gradual removal of the tube increases the current and *vice versa*. To furnish an approximate guide of the strength of the current, the tube of Duchenne is divided into ten equal parts by making file marks or by pasting a narrow strip of paper, on which the divisions have been registered, on the tube. The divisions are referred to as degrees. In the sledge induction coil of Du Bois-Raymond the secondary coil is moved bodily over the primary current. The registration of intensity is marked on a scale fastened to the apparatus, which is divided according to the size of the apparatus in 10, 50 or 100 degrees. This instrument is much more sensitive than the tube induction coil, and an exact differentiation between the various degrees is more readily obtained.

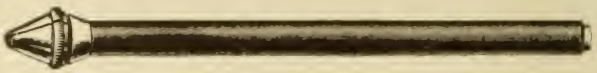


Fig. 51.—Insulated electrode holder.

The small faradic battery carries three binding posts and furnishes three definite currents. Posts 1 and 2 furnish the mild primary current, posts 2 and 3 furnish the more intense secondary current, while posts 1 and 3 furnish the strong combined currents. The metallic electrode fastened to one pole is held in the hand by the patient, while the other pole carries the conducting cord, to which a specific dental electrode is attached. Whether the respective electrode is attached to the positive or the negative pole is of no consequence as the direction of the passing current does not influence the results. The dental electrode may consist of a piece of hard rubber in the form of a penholder, with a piece of German silver wire passing through its body. A socket is left at each end for the attachment of the conducting cord and the copper point. The latter is slightly roughened to carry a small piece of wet cotton; it may be bent to any desired angle and sterilized in an open flame. A hard rubber electric needle holder with contact switch (interrupter) as used by the dermatologist for the removal of hair and which may be procured from surgical supply houses makes a most serviceable dental electrode.

The Action of the Faradic Current on the Pulp.—The diagnosis of the condition of the pulp for clinical purposes resolves itself into the recognition of hyperemia, acute simple and suppurating inflammation and death of the organ. The action of the electric current on a sound tooth calls forth a definite sensation which is the physio-

logic expression of the normal reaction of a patient to electric stimulation. The strength of the current needed for this purpose varies with the individual. The reaction manifests itself in a peculiar tingling sensation, but *not in pain*. This point is known as the "irritation point," or the threshhold of sensation. After having established the irritation point in a sound tooth of the patient, and after having expressed it in figures from the markings on the tube of Duchenne, it is a simple matter to distinguish a diseased or a dead pulp from a healthy pulp. By comparing these figures a reliable clue for the diagnosis of an existing disease of the pulp is furnished. The following scheme may serve as a guide for making a diagnosis:

1. The normal pulp responds to the faradic current at its specific irritation point.

2. The hyperemic pulp responds to the faradic current slightly below the irritation point.

3. The inflamed pulp (acute simple pulpitis) responds to the faradic current always *below* the normal irritation point. The more intense the inflammation, the more ready the response to the current.

4. The suppurating pulp (acute or chronic suppurative pulpitis) responds to the faradic current *above* the normal irritation point. The more intense the suppuration, the less ready the response to the current.

5. The dead pulp (necrosis or gangrene) does not respond to the current, not even to its full strength.

To illustrate this diagnostic scheme by figures as obtained from measuring with the tube of Duchenne, the following data may serve as examples:

	Degrees	Diagnosis.
1. Upper first incisor . . .	3.5	Normal irritation point.
2. Upper right first premolar .	3.0	Hyperemia (tooth shows a slight carious defect).
3. Lower right first molar . .	1.5	Acute pulpitis (tooth shows a deep carious defect).
4. Lower left second molar .	7.5	Suppurative pulpitis (the tooth has a large compound amalgam filling).
5. Upper left second premolar.	No response from current.	Dead pulp (the tooth has a large cement filling).

Technic of Applying the Faradic Current to the Tooth.—The metallic hand electrode is held by the patient, or a wet cork or felt electrode is fastened to his wrist. The other pole carries the dental electrode, which is manipulated by the operator. The current is started at its lowest voltage—*i. e.*, the tube of Duchenne is completely pushed over the core, or the sledge is started at zero. The irritation point of the patient is now obtained by holding the dental electrode charged with a piece of moist cotton against any of the apparently sound teeth. A tooth corresponding to the one under observation

is preferably selected for this purpose. The wet cotton of the electrode is placed near the center of an accessible surface of the selected sound tooth, but always away from a present filling. The tube is now gradually withdrawn until slight, but distinct, sensation is felt by the patient. The sensation must never be expressed as pain. The number on the scale of the tube is read and the same maneuver is repeated on the suspected tooth. The deduction obtained by comparing the two readings furnishes the irritation point of the diseased tooth. The average irritation point is not the same for every tooth and for every patient. A layer of thick enamel on a heavy body of dentin requires a stronger current and *vice versa*. Consequently, the irritation point in the young is usually lower than in old individuals. The respective condition of the nervous system of a patient also influences the response to the current; a disturbed psyche is usually more sensitive to electric stimulation than a normal condition. If the electrode is placed on or very close to a metallic filling in a vital tooth, the response is very pronounced, and even painful, as compared with the same amount of current passing through a tooth without a metallic filling. This is also true if the electrode is placed on a thin shell of enamel which covers a metallic filling. The severity of the shock depends on the nature of the filling. All filling materials with the exception of gutta-percha are better electric conductors than enamel.

The tooth under observation must be dry, and not in too close contact with its neighbors, as the current may switch to an adjoining tooth. The close proximity of large contour fillings or metallic crowns deserve special care. In such cases the rubber dam or strips of the dam placed between the adjacent teeth is necessary for insulation. The electrode must not be placed too near the gum line, or the gum tissue may react before the pulp is reached. The sensation felt on the gum is quite different from that in the pulp. It is not acute, but manifests itself as a tickling or crawling sensation. Devitalized teeth which carry metallic fillings will also react if the electrode is placed on or near the fillings; they will not react if the electrode is placed on sound enamel provided that the root filling consists of gutta percha. If the root carries a metallic post a prompt shock is felt from the current. If a present filling reaches the gum line a very quick and painful response is experienced, even from a mild current, when placed in contact with the filling. The absence of enamel acts somewhat similar to the presence of a filling. A shock is usually produced when the current is placed on exposed dentin which must, therefore, be avoided. A tooth with a dead pulp, but with a sound crown, may also react to the current if an acute pericementitis is present. Usually, however, a somewhat stronger current is required than that which is necessary to establish the normal irritation point. In multi-rooted

teeth the pulp may be dead in one canal and highly inflamed in another canal. In such cases a reaction similar to that obtained from suppurative pulpitis is usually observed.

Disturbances involving the general nervous system, especially locomotor ataxia, manifest themselves in the teeth by a more or less complete analgesia of their pulps. In such cases, although the pulp seems to be apparently normal, the current produces no response.

The examination of the pulp by means of the faradic current requires a thorough mastering of the many details connected therewith. The practitioner can best familiarize himself with the current by testing the instrument on himself or on an experimental patient. The teeth, gums, lips and tongue are organs which should preliminarily be tried. Before testing a tooth it is always advisable to establish, if possible, the irritation point in a corresponding sound tooth. The difference of the recorded figures furnishes the base for its diagnostic utilization. It is understood, of course, that no therapeutic measures (drugs) have been previously applied to the tooth under consideration or to the general system. Their presence would materially influence the reaction of the current. Morphin, for instance, if administered in average doses, will reduce the reaction of the current from three to four degrees below the normal irritation point, while chloral hydrate in 15-grain (1 gm.) doses acts within three to four minutes, and reduces the scale two to three degrees.

All of the enumerated methods of diagnosing the respective state of health of the dental pulp should be looked upon as helpful aids, but by no means as absolute proofs. The results obtained with the faradic current are so very superior to all the diagnostic procedures that its use for such purposes deserves to be highly recommended. Men with only limited experience in employing this apparatus have passed premature judgment regarding its merits and its shortcomings. Incompetence, as displayed in the utilization of a certain apparatus, does not necessarily indicate that the instrument is at fault.

The direct diagnosis of an inflamed pulp by the various methods as outlined above should always be supplemented by a *differential diagnosis* in regard to a concomitantly existing disturbance of the peridental membrane of the affected tooth or of a general disease. This examination constitutes the absolutely necessary adjunct for a final diagnosis. The differential diagnosis of the pulp and an existing general disease is discussed under Secondary Pulpitis. The appended scheme may serve as a basis for a differential diagnosis of the inflammatory diseases of the pulp and the peridental membrane:

DIFFERENTIAL DIAGNOSIS OF INFLAMMATORY DISEASES OF:

The Dental Pulp.	The Peridental Membrane.
The pain is of a sharp, lancinating character; in the earlier stages it is distinctly paroxysmal. On assuming a recumbent position, or with excitement or fatigue, the pain usually increases.	The pain is localized, dull, steady, boring or throbbing in character; it is not paroxysmal, nor increased by a recumbent position. Pain remains more or less constant without much reference to external conditions.
The tooth is exceedingly sensitive to thermal changes; in its inceptive state cold; later on heat intensifies the pain.	There is little reaction to temperature changes; cold may give relief, while heat does not materially affect it.
There is no swelling of the soft tissues about the tooth, and no tenderness to pressure.	Pressure at first usually relieves the pain, later it is intensified. In the later stages swelling is common.
The tooth is not elongated nor does it strike first in occlusion.	The tooth is raised in its socket and strikes before any of the others occlude.
At times it is quite difficult to determine exactly which tooth is affected as the pain induces reflex symptoms in other teeth and tissues.	The diseased tooth is readily located; the pain is steady in degree and in its position. No reflex symptoms are observed.
Percussion is negative.	Percussion induces pain.
It is possible to bite upon the tooth and to use it in mastication without any special sensation if thermal extremes be avoided.	The tooth is very sore to the touch; occlusion in mastication or ordinary shutting of the teeth produces pain irrespective of thermal changes.
The tooth usually shows a carious defect.	Carious defects, as such, are of no consequence.
Swelling of the mandibular lymph nodes is not observed.	The mandibular lymph nodes A, B, C or D are swollen, tender and painful on pressure.

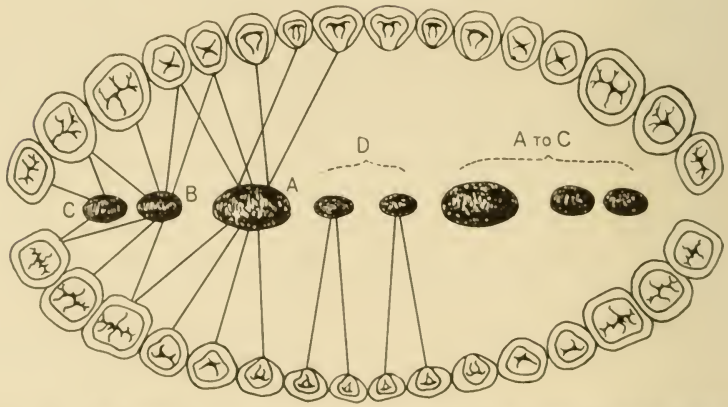


FIG. 52.—Schematic drawing of the relationship of the submaxillary and the submental lymph nodes to the teeth of the upper and the lower jaw. *A*, *B* and *C* = submaxillary lymph nodes; *D* = submental lymph nodes.

The final diagnosis is based on the observations obtained from a careful comparison of all the elucidated facts and usually leads to a *diagnosis by exclusion*. Its interpretation results in the prognosis, and the final goal of all clinical procedures, the treatment of the existing disease.

CHAPTER VI.

HYPERSENSITIVE DENTIN.

Hypersensitive dentin may be defined as a *state* in which the exposed dentin of a vital tooth is painfully responsive to mechanical, chemical, thermal or electric irritation. The primary cause must be always attributed to its exposure to an irritant. Absence of enamel or otherwise pathologically exposed dentin are the necessary initial conditions essential for its occurrence. Enamel, which normally protects the dentin of the crown, may be absent as a sequence of incomplete calcification or it may be lost through pathologic processes, *i. e.*, caries, erosion, abrasion or trauma, while the exposed dentin of a tooth root is primarily brought about by premature or senile atrophy of its protective alveolar process and its cementum.

Pathology.—Before entering upon a discussion of the pathology of hypersensitive dentin, the anatomy and physiology of normal dentin should be briefly recalled. Dentin is made up of about 72 per cent inorganic salts, about 10 per cent water and an organic colloidal matrix constituting the remaining percentage. The dentin is traversed by a very large number of tubules measuring about 1.5 to 5 μ in diameter, and radiating from the pulp cavity more or less wave-like toward the periphery, where they branch off, forming a deltoid network. Roemer has counted from 25,000 to 30,000 dentinal tubules within the area of 1 sq. mm. The tubules are filled with lymph and with the protoplasmic processes of the odontoblasts, as originally described by Koelliker, and they are known at present as Tomes' fibers. These fibers are structureless threads and are continuous through the full length of the tubules and their branches. Physiologic normal dentin has no sensation; its vital protoplasm transfers tactile impressions, thermal changes and chemical or electric irritation to the pulp.

The so-called innervation of dentin is still a much mooted question. Professor Hopewell-Smith interprets its present status as follows:

It is an interesting and important fact that it has not yet been proved to the entire satisfaction of all observers that nerve fibers exist in the dentinal tubes. Thus, there is a wide difference in men's views as to the innervation or non-innervation of the dentin. One school of belief, headed by Boll, Morgenstern, Roemer, Depen-

dorf, F. Boedecker, Fritzsch and Howard Mummery endeavor to explain sensitiveness to the occurrence of peripheral nerve fibers in the dentinal tubules, while the other school denies the existence of nerve fibers in this tissue. Among the names of those included in this group are those of Retzius, Koelliker, Tomes, Huber, Walkhoff, Gysi and Hopewell-Smith. By persistent search in teeth of mammals and reptiles no definite nervous system has been demonstrated, the nerve fibers terminating in arborizations around the odontoblasts on the surface of the dental pulp.

Impulses are carried through the dentin to the pulp, *via* the contents of the dentinal tubules, *i. e.*, dentinal fibrils—the peripheral processes of the odontoblasts—and lymph. There is an abundance of protoplasm in these innumerable channels. Members of the second school of thought are divided in their views as to the causes of sensation. The hylopathist ascribes it to abnormal movements of the molecules of the dentinal fibrils while the others claim that demarcation currents, convection and osmosis are responsible for pain. All, however, are agreed that the cerebrospinal nervous system has no share in its production beyond that in the dental pulp.

Personally, the writer is in full accord with the concepts of the second school of histologists, *i. e.*, the non-innervation hypothesis of dentin. From a pharmacologic point of view he is able to furnish sufficient data to substantiate this assumption. Basing his own conception upon this hypothesis, he assumes that hypersensitive dentin denotes a state in which the contents of the dentinal tubules are pathologically altered. This change is brought about by external physico-chemical influences which interferes with surface tension, adsorption (inbibition) and diffusion. All processes are closely allied phenomena.

According to Gibbs' law, all substances which lower the surface tension of a solvent become more concentrated in the surface film than in the interior. It is a phenomenon which depends upon the increase of attraction of the molecules in the surface film for one another and puts the film under pressure. Thereby a hydrostatic pressure is created which materially increases the normal osmotic pressure. As a rule, inorganic neutral salts, and many sugars very slightly raise the surface tension, whereas acids, bases and most organic substances lower the surface tension. The colloids concentrated in the surface film become very viscous, finally forming a membrane insoluble in water. Colloidal solutions readily adsorb water and dissolved salts from the surrounding medium. The adsorption of water increases proportionally with the concentration of the salt solution to a certain point and thereby an increase in the internal pressure of the colloidal solution is obtained.

Surface tension is constantly trying to reduce itself; in a uniform

fluid this is impossible while in a mixture consisting of two or more substances, which in themselves possess different surface tension, the lighter fluid has a tendency to collect on the surface of the more tense fluid. Under the influence of these different forces, dynamic equilibrium is established within a certain time.[1] These various factors favor mechanical adsorption or imbibition, reduction of surface tension, increase of solubility under pressure and compressibility of water. It is interesting to observe that alcohols, fatty acids, esters and many organic solvents are readily adsorbed.

Chemical adsorption is of less interest in this connection. The most important factor which influences adsorption is the ion concentration of a fluid. The equilibrium of a phase relative to its ion concentration is controlled by the law of mass action.

The relative viscosity of a fluid plays a most important role. If the surface of a solution adsorbs a dissolved substance the viscosity of its surface may markedly increase. As a consequence, albumins, soaps, saponins, dyestuffs, etc., form surface films which materially interfere with the diffusibility, as compared to pure water of dissolved substances.

When the colloidal contents of the dentinal tubules become exposed to the fluids present in the oral cavity, their surface tension become altered by adsorption and diffusion in accordance with the above enumerated physico-chemical processes; they become overdistended and thereby exert pressure upon the underlying odontoblastic cells. The fluids in the tubules cannot be compressed, as water possesses no elasticity; it represents a rigid column which transmits pressure in the form of motion undiminished in all directions. Any additional pressure which is exerted upon the overdistended surfaces is at once transmitted to the nerve filaments located at the surface of the pulp (the plexus of Boll), *i. e.*, the anatomic threshhold of sensation.

Cutting the enamel does not produce painful sensation. As soon as the amelo-dentinal junction is reached marked pain is usually experienced by the patient. Beneath the amelo-dentinal junction are located the interglobular spaces of Czermak which are completely filled with semifluid protoplasm. Pressure and heat produced by the revolving bur upon a relatively large surface area of fluid in this region are quickly transmitted to the pulp and hence pain is felt. Within the area of dentin which lies beyond this borderline zone sensation again is lessened until the advancing bur reaches within close proximity of the pulp. In carious dentin excavation of the zones of complete disorganization and of decalci-

[1] Decrease in surface tension is readily demonstrated by the following simple experiment: To 100 cc of absolute alcohol contained in a graduated cylinder add 100 cc of distilled water. After equilibrium is established the mixture measures only about 192 cc at room temperature.

fication does not produce sensation because the contents of the tubules are destroyed. As soon as the zone of turpidity is reached again marked pain is manifested. Here the contents of the exposed dentinal tubules are subjected to intense irritation brought about by acidity and other products of bacterial metabolism. The surface tension of the fluids in the tubules is markedly altered, hence the quick response to pressure and thermal influences. Below this zone of turpidity, the "translucent zone" of Tomes is observed in chronic caries. This translucency of dentin is the product of a vital reaction. The chronic irritation of the odontoblasts causes the pulp to promptly respond by depositing adventitious dentin within the lumen of the tubules which necessarily lessens their

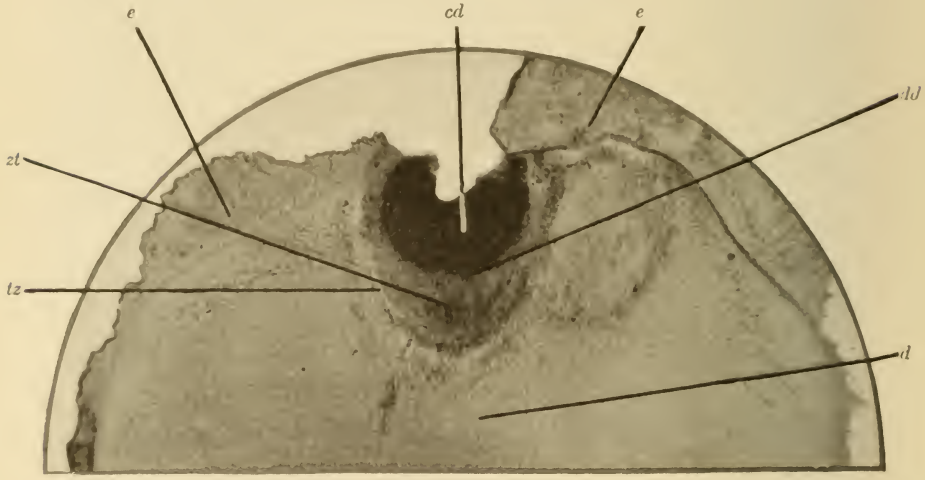


FIG. 53.—Sections of a carious tooth: *e*, enamel; *d*, dentin; *cd*, carious dentin; *dd*, decalcified dentin; *zt*, zone of turbidity; *tz*, translucent zone. (Photomicrograph by Hopewell-Smith.)

diameter in varying degrees or even produces complete obliteration. Hence, a smaller surface of the tubular contents is exposed to the advancing bur and, consequently, lessened sensation is felt. The gradual reduction of surface area of the dentinal tubules is a physiologic process in the life cycle of a tooth, hence sensitiveness diminishes with advancing age.

Mechanically abrated teeth or those subjected to the as yet little known process of erosion are rarely hypersensitive in the latter states. Abrasion and erosion are usually intensely chronic processes, hence their very slow progress offers to the irritated odontoblasts sufficient time to deposit adventitious dentin within the tubules and thereby protect the underlying pulp from further

irritation. Sections of mechanically or chemically abrated teeth containing living pulps always show a translucent zone.

As stated above, the process of removal of enamel of a sound tooth by cutting and grinding, if done under proper precautions to avoid undue heat, is usually not painful. The freshly exposed dentin is relatively free from sensation. Within a short lapse of time, however, usually within twenty-four hours, this exposed dentin is excruciatingly hypersensitive. The exposure of the contents of the tubules to the fluids of the mouth as explained above, changes the surface tension so as to cause pronounced irritation of the odontoblastic cells.

FIG. 54.—Silver nitrate applied to carious dentin upon the living tooth in the mouth. Low power.

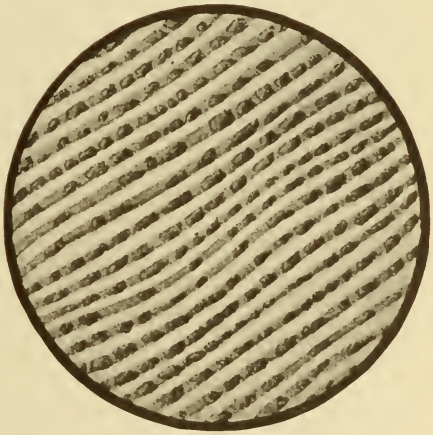


FIG. 55.—Magnified section of Fig. 54.

Dehydration of the overdistended tubules by physical means relieves the hypertension; consequently, such an agent as warm air reduces the sensibility. Alcohol, potassium hydroxid or similar hygroscopic chemicals act synchronically as dehydrants and caustics. Self-limiting caustics as silver nitrate, superoxol, etc., superficially destroy the vitality of the protoplasmic fibers, and they protect the contents of the tubules by solid plugs of precipitated albumin. The disturbing elements are thereby permanently excluded from reaching the dentin. The disarranged equilibrium of the tubular fluid readjusts itself in a short time and consequently hyperesthesia is relieved. A substantial illustration of this fact is furnished by immediately protecting artificially exposed dentin with a coating of silver nitrate or a temporary cap set with gutta-percha and *not* with an irritating cement. Such dentin will exhibit no particular sensation at any time after the operation.

Any general condition which lowers the normal psychic reaction of a patient as a whole naturally also influences the reactivity of the tooth pulp. Therefore, such disturbances as acute nasal catarrh, influenza and other infectious diseases, exanthematous fevers, increased intradental blood-pressure, menstruation, anemia, general debility or certain neuropathic conditions, as neurasthenia, may leave their imprints upon the pulps in the form of congestive hyperemia and, consequently, any irritation of the exposed dentin of a tooth under these conditions is prone to exhibit indirectly excessive sensibility.

From the above discussion of its pathology, the writer concludes that hypersensitive dentin designates a state of irritation of the odontoblasts of the vital pulp. Irritation is produced only by external agents, *i. e.*, physico-chemical processes induce changes in the surface tension (hypertension) of the exposed contents of the tubules. The increased reactivity of the disturbed equilibrium transmits any additional physical or chemical impulse at once *via* the Tomes' fibers to the congested odontoblastic cells covering the pulp and which are in direct contact with the nerve fibers of the plexus of Boll, *i. e.*, the anatomical threshhold of sensation.

Symptoms and Treatment.—The principal subjective symptom of hypersensitive dentin consists in more or less severe pain which is usually elucidated by marked temperature changes, chemical or electric irritation or mechanical interference of the exposed dentin surface. Thermal irritation is quickly and profoundly manifested by cold; 68° F. (20° C.) or below leave painful impressions, while heat, even up to 140° F. (60° C.) are often unnoticed. Chemical irritation results principally through an increase of surface tension (osmosis) or by dehydration. Many foodstuffs, especially condiments, contain substances belonging to this group, among which fruit acids, salt and sugar play a most important role. Occasionally, electric shocks arising from contact of opposing metals, *i. e.*, an amalgam filling and a gold crown, etc., elicit painful impressions. Mechanical irritation brought about by use of instruments during dental operations, and rarely from the application of external forces, is probably the most common source of interference with the exposed dentin surface. The manifested pain is *not* continuous; it merely lasts as long as the irritant is present. The patient is always able to point to the affected tooth. Inspection may reveal a carious or other defect of its crown, an exposure of its root or frequently an incomplete union at the periphery of the enamel and cementum at its neck. The thermal test with hot and, especially cold water, or pressure exerted by an instrument placed upon the exposed dentin surface, is very pronounced. Changes in the color of the tooth, percussion, palpation and roentgenogram are negative. Hypersensitive dentin offers good chances

for conservative treatment; under proper management it may readily be eradicated.

The rational principle of treatment should be based on the recognition of its pathologic cause, *i. e.*, hypertension of the contents of the dentinal tubules. Any method or means which favors the readjustment of the altered colloidal equilibrium and prevents further irritation of the exposed dentin surface is useful for the purpose.

In general the remedies employed should conform to the following requirements:

1. The remedy must not injure the organic or inorganic constituents of the tooth.
2. The remedy must not permanently interfere with the welfare of the pulp.
3. The administration of the remedy must not require a complicated instrumentarium.
4. The pharmacologic action of the remedy must be exhibited within a few minutes.
5. The remedy must be readily applicable to all classes of cavities with regard to their location.
6. It must not produce pain.
7. Permanent discoloration of dentin must not occur.

For convenience, we may divide the applied remedies into:

A. Physical and chemical procedures:
   1. Keen-edged instruments.
   2. Caustics.

B. Local and general remedies:
   1. Local anesthetics and sedatives.
   2. General anesthetics and sedatives.

Sharp Instruments.—The superiority of sharp instruments as compared with dull ragged-edge tools when working upon living tissue is generally recognized by every-day experience. Sharp excavators cut without much pain when employed with a definite, precise movement at right angles to the long axis of the tubules. Dull engine burs produce heat by friction and by being held in contact with the cavity wall too continuously. They should not only be sharp, but run at high speed and allowed to touch the surface very lightly as they revolve. A thin coating of vaselin further reduces undue friction.

Caustics.—Caustics are substances which destroy living tissue by virtue of their coarse chemical or physical action. This action may manifest itself by abstracting water from albumin, by dissolution or precipitation of the albumin, by oxydation or by substitution. Caustics which are employed for the purpose in view are principally dehydrants and albumin solvents or precipitants. Alkalies containing hydroxyl groups—KOH and NaOH—are very powerful

albumin solvents and they are not self-limiting. The albumin precipitants are primarily represented by the metallic salts, by certain organic compounds as phenol, alcohol, etc., and by heat. Mineral acids should not be applied on living tooth structure for such purposes. The precipitates obtained by metallic salts differ widely in regard to their density; silver nitrate, for instance, produces a dry dense scab, while zinc chlorid combines with the albumin to form a loose flocculent clot.

As we have stated above, hypertension of the contents of the dental tubules is the primary cause of hypersensitive dentin. The removal of this tension will necessarily interfere or prevent the transmission of impulses; hence the simplest and most logical method of reducing hyperesthesia of dentin for the purpose of excavating is to dispel the moisture from the tubules. It has been found that desiccation of a cavity by subjecting it to a current of warm air in conjunction with absolute alcohol will bring about a condition of immunity to sensation in proportion as such desiccation is thorough or partial.

Benzyl alcohol, also known commercially as phenmethylol, possesses the combined properties of acting as a local anesthetic and a dehydrating agent. It may be employed in pure form or in the following combination:

Benzyl alcohol	50 parts
Chloroform	30 "
Alcohol	20 "

To best accomplish desiccation of the dentin the rubber dam should be adjusted to the tooth and the greater portion of the carious mass is carefully removed with spoon excavators; the cavity should be bathed with absolute alcohol[1] and then subjected to a stream of warm air applied in some convenient manner. The ordinary air syringe or chip blower may have its point heated in a flame, and then by forcing the air in the bulb slowly through the tube a jet of warm air will be delivered in the cavity. By holding the nozzle of the syringe at the proper distance, and having learned by experience how much heat to apply, one can often inject a current of air into the cavity at nearly the same temperature as that of the tooth; but if the air when it reaches the cavity should be either perceptibly above or below the proper temperature pain will be produced. In some warm-air syringes the tube is provided with a hollow receptacle somewhere along its length, which, when heated, raises the temperature of the air within it before being directed into the tooth cavity. Neither of these methods is

[1] Absolute alcohol for this purpose may be prepared by adding either ½ ounce anhydrous copper sulphate or an equal quantity of well-burned unslaked lime to 3 ounces of commercial alcohol.

at all exact, and they are therefore liable to produce more or less pain in the act of dehydration. A better plan is to employ a syringe in which a coil of fine platinum wire is contained within the orifice; this coil is connected by wires through the body of the syringe with a source of electric current; in operation the resistance encountered by the current of electricity passing through the platinum coil heats it and maintains a steady temperature. Air forced over this coil and through the nozzle, especially air supplied from a receiver and under pressure that can be controlled, may be heated to a temperature that will approximate very closely that of the tooth, and therefore produce little or no pain. If the air passing from the nozzle of the syringe should be too warm it can be modified by holding it a little farther away from the tooth, or if not warm enough, more heat will be delivered when it is held in closer proximity.

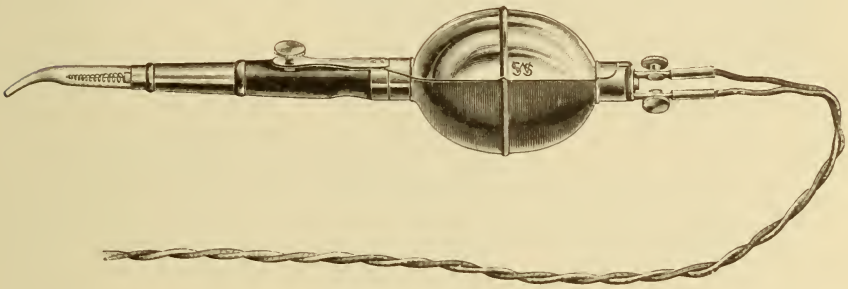


Fig. 56.—Electric warm-air syringe. (Guilford.)

An instrument of this character with a compressible blub instead of an air supply from a receiver is represented in Fig. 56. The operation of desiccation should not be hurried; time must be allowed for raising the air to a suitable temperature, so as to cause as little pain as possible. In addition, the operation should be continued until the dentinal walls of the cavity have become perceptibly lighter in color, indicating that they have been robbed of their moisture. If desiccation is not carried to this point it will fail in its effectiveness; but, if the moisture has been removed from the dentin to a considerable depth, as it may be if desiccation be sufficiently continued, sensitiveness will have become nearly or entirely obliterated. Whether we depend entirely upon dryness to relieve hypersensation of dentin or not, it should always be resorted to, for it proves a most valuable preliminary means where it is to be followed by medication of any kind.

Caustic alkalies are preferably applied in the well-known form of Robinson's remedy which is composed of equal parts of potassium

hydroxid and crystalline phenol, forming potassium phenate when triturated together in a warmed mortar, with the addition of a small quantity of glycerin to render it plastic. It should be prepreserved in well-stoppered bottles. Another serviceable application consists of an intimate mixture of 1 part of crystalline sodium carbonate and 5 parts of potassium carbonate. It produces a pasty mass which must be kept in a tightly corked bottle. A small quantity of these compounds applied to the previously dehydrated painful dentin surface and rubbed into it with a warm burnisher will often prove to be of benefit. The caustic and dehydrating effect of these agents combined with the warm air blast lowers hyperesthesia markedly; their benumbing action, however, is only superficial and they have to be repeatedly applied as the preparation of the cavity progresses.

Albumin precipitants are principally represented by silver nitrate, zinc chlorid and phenol. The silver salt acts very superficially and very slowly. Incidentally, by combining with the chlorin present in the albumin and in the presence of light it pro-

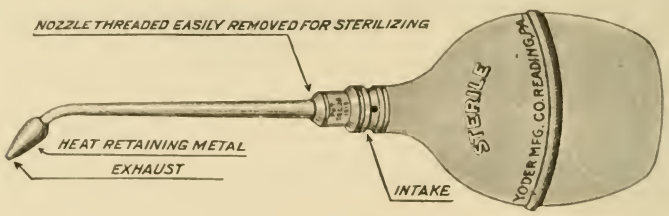


FIG. 57.—Sterile warm-air syringe.

duces a jet-black discoloration of the involved dentin. On all exposed dentin surfaces, especially on the exposed roots of the posterior teeth where the resultant color is no objection, it is an admirable desensitizer. It is best applied as a freshly prepared saturated aqueous solution. Other silver salts are of less value in this connection, as they are less caustic. It should be remembered that the pharmacodynamic action of silver nitrate depends upon the precipitation of albumin by the nitric acid ion and *not* upon the silver ion. The latter merely combines with the albumin, forming a complicated double salt, *i. e.*, silver-albumin chlorid, which in the presence of light is partially reduced to a black oxid. Zinc chlorid is an admirable desensitizer; its application in crystal form or as a saturated solution is somewhat painful on account of its acid reaction. As it is not self-limiting, it should not be applied into deep-seated cavities on account of the danger of pulp irritation at the time of its application or subsequently. Liquefied phenol does not penetrate deeply into tooth structure and may be safely applied to cavities of any depth. When applied into a

dehydrated cavity in conjunction with the warm air blast it produces quick and marked superficial benumbing effects, hence it is widely employed for this purpose. The addition of local anesthetics to phenol for this purpose, *i. e.*, cocain, etc., is an irrational procedure.

Within the last few years Buckley has lauded dry formaldehyd (trioxymethylene) in the form of a paste as: "A new, safe and reliable remedy for hypersensitive dentin." This empirically compounded paste contains approximately 35 per cent of dry formaldehyd rubbed up with vaselin and a few minor substances of no direct value. International dental literature of the last decade is filled with references relative to the use of formaldehyd as a

FIG. 58.—Photomicrograph of a longitudinal section of a healthy tooth treated in the mouth with a protoplasm poison and a vital stain. The pulp was vital as ascertained by the electric current. At *a*, a cavity was drilled and a small quantity of formaldehyd desensitizing paste containing 1 per cent of methylene blue was sealed into it for three days after which time the electric current revealed a dead pulp. Between *a* and *b* the bluish-green stain indicates the path of the formaldehyd on its way through the dentinal tubules into the pulp, at *c* the coronal portion of the latter is also stained. As methylene blue only stains dead dentin it closely followed the formaldehyd on its devitalizing passage through the tubules into the living pulp.

desensitizing agent and all writers, except Buckley, agree that it is a most dangerous agent for this purpose, as it will injure and, in most instances, kill the pulp. It produces numbness of dentin in the same manner as arsenic, only acting somewhat slower. Trioxymethylene acts as a non-self-limiting caustic which penetrates comparatively quickly through any thickness of dentin. As an illustration of the intense caustic action it may be stated that in the hands of some practitioners the Buckley desensitizing paste constitutes the routine application for the purpose of destroying the pulps in deciduous teeth. The same deleterious results are obtained with the so-called "Norwegian Dentin Anesthetic." This

compound contains carpain and paucin, two alkaloids which act somewhat like erythrophlein, *i. e.*, they kill the pulp.

Occasionally protoplasm poisons are recommended for the purpose of desensitizing dentin. In many instances these drugs are erroneously referred to as caustics. A protoplasm poison should be designated as a drug which endangers, or even kills, living-cell structure without visible changes. Protoplasm poisons are not self-limiting in their action. Arsenic trioxid and, to a less extent, the alkaloids nervocidin, erythrophlein and paucin are the principal substances of this group that have been employed as desensitizing

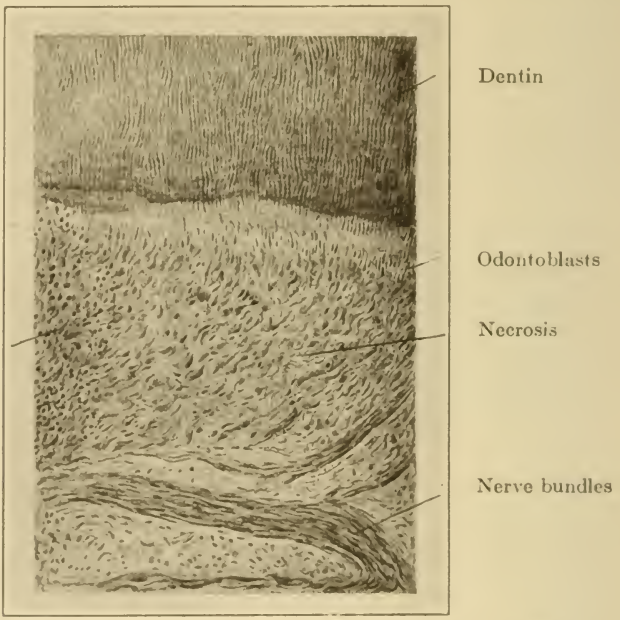


FIG. 59.—Section of a tooth treated with Buckley's desensitizing paste. Pulp shows extensive necrotic areas near point of application. (Demisch.)

agents. Arsenic, when applied even in the very minutest quantities, will usually always kill the pulp, as its action cannot be controlled. This is equally true of the above named alkaloids; they have only historical interest at present.

Local Anesthetics and Sedatives.—True local anesthetics, *i. e.*, cocain or its substitutes when applied to exposed sound dentin without pressure do not produce any pharmacologic effects. Even if sealed into a fairly deep-seated cavity in which the underlying dentin is not decalcified, no effect is obtained. Living protoplasm reacts unfavorably against the ready absorption of substances by endosmosis for two reasons: (1) The albumin molecule is rela-

tively very large and is not easily diffusible; and (2) it possesses, as an integral part of its life, vital resistance toward foreign bodies. According to Hertwig, protoplasm primarily transfers irritation and secondarily, transmits absorbed materials. Therefore, the anesthetic solution has to pass through the entire length of the dentinal fibers before the nerve tissue of the pulp proper is reached. Consequently, a certain period of time is required before the physiologic effect of the anesthetic is manifested and the period of this latency is dependent on the thickness of the intermediate layer of dentin. The migration of a protoplasm poison through dentin may be actually observed by adding a vital stain to it, as, for instance, methylene blue added to arsenic or dry formaldehyd. The time required for its passage through about 5 mm. of sound adult dentin is from twenty-four or more hours. The pharmacodynamic power of a drug depends upon its reaction with the living protoplasm through the catalytic action of ferments. The decomposition of the absorbed drug occurs comparatively quickly, usually within minutes. These observations are seen daily on injecting anesthetics or other solutions hypodermically. An average hypodermic dose of cocain is completely decomposed by the ferments of the protoplasm within the period of an hour, *i. e.*, its typical local anesthetic effect is manifested within a few minutes after the injection. The anesthesia remains at its height for some thirty minutes and from there on it diminishes until by the end of the hour fairly complete recovery of normal sensation has reoccurred. Therefore, if we apply cocain to sound dentin it is decomposed on its passage over the Tomes' fibers and *before* it reaches the threshhold of sensation, *i. e.*, the nerve plexus at the surface of the pulp and hence no anesthesia is produced. The nature of the cocain salt, *i. e.*, whether is a hydrochlorid, a nitrate or a lactate, has no bearing upon its therapeutic action. The apparent results obtained with these cocain salts must be attributed to the preliminary dehydration, protection of the exposed dentin by a temporary filling, etc., and *not* to its therapeutic effects. This is equally true in regard to most of the heterogeneous mixtures of cocain with other substances, as, for instance, potassocain, vapocain, etc. Again, in the widely recommended solution of cocain (alkaloid) in chloroform and ether the cocain base plays no part. The apparent result obtained are produced by the process of "evaporating to dryness" and thereby obtaining a marked reduction in temperature which is the obtundent factor. When cocain or its substitutes are forced into the living protoplasm of the unobstructed dentin tubules under pressure, its anesthetic action is manifested within a few minutes. The vital resistance of protoplasm is readily overcome by comparatively slight force which quickly transfers the anesthetic solution by an increased osmotic interchange to the surface of the pulp.

The phenomenon is to be explained as an anesthesia obtained by intimate contact under pressure, either mechanical or by electromotive force (cataphoresis). The pulp of a tooth and consequently the dentin may be completely desensitized by any one of the well-known methods of contact anesthesia, by using hand pressure or that derived from a dental hypodermic syringe or some other more complicated apparatus or by electricity.

Electrical Endosmosis.—Some years ago cataphoresis was much lauded for the purpose of desensitizing dentin. This process consists in placing a concentrated solution of cocain on cotton in the sensitive cavity, and having it carried along the dentinal tubules toward the pulp by means of a galvanic current, *i. e.*, by electromotive force. A battery is employed with the negative electrode, the cathode, inserted in the cavity, and the anode placed upon some part of the patient's body, as the hand, etc. The current carries the cocain *via* the Tomes' fibers into the pulp and anesthetizes it. While in this condition, which usually lasts for an hour or more, the tooth may be worked upon without any pain. For a while this method met with great favor because of the perfect results obtained, but it was found to be a very slow process, requiring a cumbersome apparatus and often consuming more time than the operator had at his command, and occasionally requiring a second application in order to produce complete anesthesia, hence at present it has been largely discarded.

Of the numerous essential oils which have been suggested as obtundents of dentin, oil of clove stands out prominently. Its pharmacologic action depends on the presence of eugenol, an unsaturated aromatic phenol. The basic constituent of eugenol consists of para-amino-benzoic acid, a body which as such does not exhibit any marked therapeutic effects. Its methylester, anesthesin is an efficient local anesthetic; however, it is only slightly soluble in water. Einhorn and Uhlfelder, taking anesthesin as a base for their synthetic research, finally succeeded in preparing para-amino-benzoyl-diethyl-amino-ethanol, commercially known as novocain or procain, which at present is the most efficient substitute for cocain.

Essential oils, in general, possess marked penetrating power. However, upon a fairly thick layer of sound dentin they are of little value when employed as obtundents. A different pharmacodynamic action is observed with arsenic. Arsenic trioxid, As_2O_3, in the presence of certain ferments of living protoplasm, *i. e.*, oxydases and catalases, is changed to the pentoxid, As_2O_5, which again is quickly reduced to the trioxid. This perpetual oxidation and reduction within the protoplasm of the cell causes a violent oscillation of the molecule of active oxygen and thereby its therapeutic effect is manifested. The metalloid arsenic merely

plays the role of an autoxidizer. The presence of the absorbed arsenic can be detected in the tissues by chemical analysis, that of absorbed cocain cannot.

Among the local sedatives refrigerant anesthetics should be mentioned. These agents lower the temperature, diminish sensation and reduce the volume of the parts to which they are applied.

Physically reducing hyperesthesia of dentin by the application of cold is best accomplished by employing a chemical which has a low boiling point. Pure ether (boiling point, 95° F. [35° C.]), free from water, produces good results. Certain other hydrocarbons possess similar properties in varying degrees, depending on their individual boiling point. Pure ethyl chlorid (boiling point, 55° F. [13° C.]) is best suited for our purpose, as it lowers the temperature of the tissues sufficiently to produce a short superficial anesthesia in a few minutes. Too rapid cooling or prolonged freezing produce deep anesthesia, but such procedures are dangerous; the circulation in the pulp may be cut off so completely as to produce death. Liquid nitrous oxid, liquid carbon dioxid and liquid air, all of which have boiling points far below zero, are recommended for such purposes, but they require cumbersome apparatus and some of these agents are extremely dangerous to handle.

In general, it should be stated that the application of cold for the purpose of obtunding hypersensitive dentin is a barbarous procedure. The initial pain produced by the cold is in many instances most intense and much more pronounced than that experienced by cutting the untreated dentin.

Indirectly, hyperesthesia of dentin may be completely eliminated by locally blocking the sensory nerve fibers leading into the pulp of the respective tooth. Any one of the well-known methods or combination of methods, *i. e.*, infiltration and conduction anesthesia, are available for this purpose. On an average, most satisfactory results in a single tooth are obtained by using the pericemental injection provided the pericementum is sound.

The paralyzation of the central end-organs in the brain by a general anesthetic will naturally also anesthetize all the tooth pulps. Nitrous oxid is possibly more often used for this purpose than other anesthetic agent. The much lauded "analgesia" of a few years ago was, as might have been expected, a failure. With the improvement in the various methods of local anesthesia, general anesthesia for this specific purpose has lost much of its former significance.

The control of hypersensitive dentin by the administration of narcotics or sedatives is rarely called for. Of the general sedatives, the bromids are usually recommended. Large continuous doses are required to manifest their action as they impair the perception of sensory stimuli only to a very mild degree. Average doses of

morphin require at least one-half hour before a depression of the sensory impulse is manifested, while chloral hydrate shows a marked lowering within ten to fifteen minutes. Morphin-scopolamin administered hypodermically causes most pronounced general narcotic effects and, of course, marked lowering of the sensory reaction of the pulp.

Sensation in a tooth may be experimentally measured by passing a weak electric induced current through it and the above data are based upon observations obtained by such measurements.

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CHAPTER VII.

THE EXPOSED HEALTHY DENTAL PULP.

The modern treatment of a wound from a surgical point of view centers about two primary objects—the removal of all necrosed tissue and establishing asepsis. Clinically, all wounds must be looked upon as being infected. If the infection is not restricted to the surface, but has penetrated into the deeper structures, drainage and antiseptics are employed.

An exposed healthy pulp, which in reality is a wound, must always be looked upon as being infected. In conformity with the difficulties as elucidated in a previous chapter regarding the treatment of an infected pulp the normal surgical aspect of the operation is completely upset. The prime requisite for any interference with the dental pulp is based on a thorough knowledge of its anatomic relationship to the surrounding wall of dentin, *i. e.*, a recognition of the constant change within its own body during the life cycle of the respective tooth.

In many instances the unintentional opening into the pulp chamber is avoidable. If it does occur it is primarily caused by unhappy circumstances or by an atypical structure of the pulp, and in the hands of the experienced operator these accidents are to be classed as comparatively rare occurrences. The density and *not* the color of the involved dentin is the safest guide to follow during the process of excavating the cavity. It is to be understood that the tooth is always placed under the rubber dam. Careful inspection of the cavity with sterile explorers and a magnifying glass is indispensable. Sterilization of the cavity with a suitable antiseptic lessens the danger of accidental infection. If the pulp is exposed direct access offers a greater chance for its conservative treatment than an inaccessible cavity; in the latter instance the operator should lose no time in devitalizing the pulp. If the pulp is still covered with a protecting layer of dentin an effort should be made to preserve this covering provided the diagnosis of the respective condition of the pulp justifies such a procedure. These cases are treated as outlined under "Hyperemia of the Pulp."

"Pulp-capping," as this process of applying a protective medium to the exposed pulp is technically known, will always remain, relatively speaking, even under the very best possible conditions an operation of chance. Nevertheless, the writer cannot subscribe

to the supposed authoritative statements of certain practitioners, namely, that pulp-capping is always a failure.

The practice of capping an exposed pulp was first attempted and successfully carried out by Philipp Pfaff,[1] of Berlin, in 1756, although Fauchard, in 1728, had described a method of placing a filling into a cavity in which he found an exposed pulp without, however, making a special effort to protect the delicate organ by a suitable covering. Pfaff employed a cap made of gold foil sufficiently large to cover the exposure and carefully burnished the edges over the sound dentinal wall. A suitable filling was then delicately placed over the capping. A detailed description of the practice of capping exposed pulps is recorded by Leonard Kocker[2] in 1826. This procedure, as described by the famous international operator a century ago, is so very interesting that it is here appended in outline: "I hope I may be permitted to detail my own method of operating in cases where the nerve of the tooth has become exposed, which I have practised for upward of thirteen years with much satisfaction and success. I require for this the following apparatus:

"1. A small iron wire, fastened to an ivory handle. The extremity of this wire I file to the size of the exposed surface of the nerve, and bend the wire in such a direction as to enable me to touch the exposed part of the membrane without touching any other part of the tooth or the mouth.

"2. A thick tallow candle with a large wick.

"I direct my patient to discharge all the saliva he may have in his mouth, and then to incline his head backward against the head supporter of my operating chair. I put the candle into his left hand and direct him to hold it in such a position that the flame of it may be on a level with his mouth, and about 8 inches from it. I now place myself on the right side of the patient, and holding his lips sufficiently open with my left hand to prevent the instrument from touching them, I again dry the cavity as perfectly as possible with a lock of cotton fastened to the point of the cauterizing wire. Having effected this, I throw away the cotton from the extremity of the wire and make it red hot in the flame of the candle. With the wire thus heated, I touch the exposed part very rapidly, so that its surface contracts without, however, suffering to it penetrate deeply into the nerve or to touch any part of the bony structure, as this would inevitably bring on suppuration and destruction of the whole lining of the membrane. The bleeding spot must be touched very quickly with the hot wire, which is sometimes necessary to be repeated two or three times before the parts are suffi-

[1] Abhandlung von den Zähnen des menschlichen Köpers und deren Krankheiten, Berlin, 1756.

[2] Principles of Dental Surgery, London, 1826.

ciently contracted. The wire should be perfectly red hot, for in this state the cautery acts suddenly, and almost entirely without pain, but when heated to any temperature short of that of red heat much pain and inflammation are generally produced. This operation is indeed so slightly painful that I have been solicited by my patients to repeat it, although they had required much persuasion to induce them in the first instance to suffer its application. It, however, must be performed very adroitly, and without any loss of time. To prevent the flow of saliva to interfere, the patient must be desired to close his lips, but to keep his mouth wide open, until the whole of the operation is finished, which he is capable to do for a certain time only.

"The nerve, which before cauterization has a fleshy appearance, is after this operation like a black point. I take care not to disturb this point, for if the black scar is removed a new wound will be formed and bleeding again will ensue; but I leave the future healing altogether to Nature, and only caution my patient against using such things as might interfere with its salutory operations. Having thus far removed all possible cause of future disease and irritation in order to prevent any unnecessary exposure of the nerve, by which inflammation and destruction of it might be produced, I now terminate the operation by fulfilling the third indication, that is, to protect the nerve against injurious impressions from without by filling up the cavity of the tooth with metal. Having again perfectly dried the cavity, I now take a small plate of very thin lead leaf and lay it upon the exposed nerve and on the immediately surrounding parts. I next carefully fill up the whole cavity with gold."

This very same procedure was advocated as a new venture by Szabo in 1902.[1]

TREATMENT OF THE EXPOSED HEALTHY PULP.

Definition.—A break in the continuity of the pulpal wall of a tooth and thereby esposing its pulp.

Etiology.—Exposure of a pulp may be caused by a trauma, such as a fracture of the tooth, cavity preparation, etc., or it may occur as a sequence of pathologic disturbances, *i. e.*, caries, erosion, abrasion, corrosive acids, etc. The former accidents usually reveal a sound pulp, while in the latter cases an infection of the pulp may be expected. The term "partially exposed pulp," which is occasionally employed to designate a condition in which the intervening layer of dentin is very thin, is a misnomer, as the pulp is *not* exposed. In reality all clinical exposures of the pulp are partial in their nature, as the pulp is never exposed in its entirety.

[1] Oesterreich-ung. Vierteljahresschrift für Zahnheilkunde, 1902, p. 368.

Indications for Capping a Healthy Exposed Pulp.—The indications for "capping" a healthy pulp, *i. e.*, covering of the exposed pulp with a protective aseptic material, may be summarized as follows:

1. Only perfectly healthy exposed pulps which are not, or are only very slightly, wounded offer suitable cases for this operation. A healthy exposed pulp has a pink color; an inflamed pulp appears as a bluish-red pulsating tissue, trying to protrude through the broken pulpal wall.

2. The healthy pulps of temporary or permanent teeth whose roots are not fully completed offer the best chances for capping. Pulp growth denotes functional activity, *i. e.*, physiologic hyperemia, hence such pulps exhibit marked properties of repair.

3. The pulps in the teeth of advanced adults and those pulps which show signs of degeneration are not suitable for capping. The latter usually undergo atrophic changes and are ready to succumb at the slightest provocation.

4. Pulps in fully formed teeth exposed by caries, erosion, abrasion, etc., and those lacerated by the revolving engine bur, should never be capped.

Pathology.—An exposed pulp practically always denotes infection. The control of an infection of the exposed pulp does not lie within the power of the operator; as yet, there are no remedies at our command which will eradicate an infection without producing some injury to the delicate structure of this organ. In doubtful cases the safest policy is always to devitalize at once.

The strict observations of the following prerequisites as applied to the operation of pulp-capping will govern its ultimate success:

1. A correct diagnosis.
2. Ready accessibility to the exposure.
3. Asepsis of instruments and of the field of operation.
4. Careful protection of the exposed pulp against drying out and future infection.
5. The application of a suitable aseptic capping material without the slightest pressure.

Pulp-capping Materials.—The materials advocated for the purpose of capping exposed pulps vary greatly; the more important ones are:

1. Depressed metallic disks or foil made of platinum, gold, lead, etc.
2. All dental cements with the exception of the silicates, preferably zinc oxysulphate cement (Fletcher's artificial dentin).
3. Gutta percha in substance or in solution.
4. Mixtures of zinc oxid with thymol, phenol, oil of clove (eugenol) iodoform, etc.
5. Various substances, *i. e.*, cork, asbestos, rubber dam, paper,

mica, celluloid, paraffin, glutol (formaldehyd-gelatin), etc., either as such or medicated or in combination with an adhesive.

6. Numerous proprietary preparations.

A pulp-capping material should possess the following properties:

1. It must be non-irritating.
2. It must be a non-conductor of temperature.
3. It must be readily adaptable and applicable without the slightest pressure.
4. It must be non-putrefactive.

The rationale embodied in the process of pulp-capping consists in applying an inert aseptic cover over the exposed pulp to protect it against further irritation. In due time the pulp usually slightly shrinks away from the break; secondary deposits of dentin which were supposed to take place according to former observers do not or only very rarely occur.

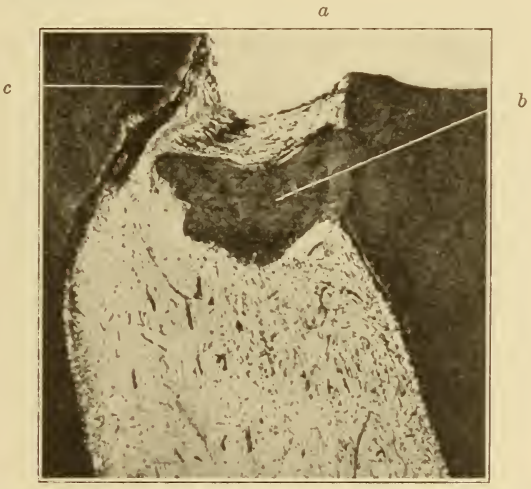


FIG. 60.—Exposed healthy pulp capped with a paste of eugenol and zinc oxide. *a*, connective tissue forming a cicatrix; *b*, splinter of dentin; *c*, newly-formed secondary tissue. (Bætwyler.)

In conformity with these requirements, the selection of a suitable pulp-capping material is a comparatively simple matter. Many of the substances advocated, especially caustic antiseptics and certain cements, are directly harmful. During the "setting" of many of the cements of the oxyphosphate type a high degree of heat—in some instances 140° F. (60° C.)—is evolved, which temperature invariably kills the pulp (p. 554). Such compounds as formaldehyd, zinc chlorid, phenol, creosote, eugenol, etc., when applied pure or in concentrated solutions, are caustics, and will in most instances destroy the pulp in due time. Even the repeated

application of such a mild remedy as oil of clove to the exposed pulp will eventually cause its death. The statement is frequently made that an exposed pulp under a formaldehyd preparation or a zinc oxychlorid cement filling will remain quiescent. A pulp treated with these substances when subsequently examined will, in the majority of cases, be found dead. The resultant necrosed tissue at some future period usually will become infected.

FIG. 61.—Placing cap over pulp exposure.

Treatment.—As stated above, strong antiseptics must be rigidly avoided; the very delicate pulp is too easily injured by most of these compounds. A pellet of cotton moistened with a weak dichloramin-T solution is placed gently over the pulp exposure to act as a protective against infection, but primarily to prevent its drying out. Irreparable shrinkage will occur if this phase of the operation is overlooked. The cavity is prepared with sterile hand instruments, the cotton pellet is removed and the cavity is dried with a fresh piece of sterile cotton, but *never* with the warm air

FIG. 62.—Watson's metal pulp caps.

blast. A bit of pulp-capping material, consisting of 2 parts of zinc oxid and 1 part of thymol,[1] is taken up with a warm suitably shaped instrument, carried to the edge of, but *not* upon, the exposure and with a warm amalgam plugger the material which has become plastic by the heat of the instrument is molded very gently and without the least pressure over the opening. The operator, especially the beginner, is cautioned against the use of a large quantity

[1] A suitable pulp-capping may be prepared as follows: One part of thymol is melted in a porcelain capsule and mixed with 2 parts of dry zinc oxid. The mixture is spread on a glass slab and on cooling it is removed and preserved in a well-stoppered bottle.

of capping material. Only enough should be employed to cover the exposure and its edges. The capping sets almost immediately. Many operators prefer to apply the capping upon a depressed metallic disk. Such disks may be obtained from supply houses, or they may be readily made by burnishing a thin piece of metal (aluminum) with a ball-shaped instrument against a stick of soft pine wood. The adjacent dentin is now sterilized with a 50 per cent thymol solution in aceton or alcohol and the cavity is then coated with a hard copal varnish. After drying, the cavity is lined with a veneer of oxyphosphate cement. The cavity may then be occluded with a gutta-percha preparation or some other temporary filling material for further observation.

CHAPTER VIII.

HYPEREMIA.

Synonyms.—Acute, active, arterial, congestive or physiologic hyperemia and chronic, passive, venous, obstructive or pathologic hyperemia of the dental pulp (*hyperemia pulpæ*).

Definition.—Hyperemia denotes the presence of an increased amount of blood in the pulp. It may be of a physiologic or a pathologic type, and partial or total in its nature. Physiologic hyperemia indicates an increased influx of blood, while pathologic hyperemia results from a retarded afflux.

Etiology.—Hyperemia of the dental pulp is the initial response of this tissue to an irritation. Only the pathologic type is of interest to us at this moment. Agents which favor sudden temperature changes, as carious defects, erosion, abrasion, etc., play a most important role. The late Dr. G. V. Black always emphasized the fact that: Hyperemia is very commonly the result of heat generated in polishing fillings, either by rapidly revolving disks, or by the vigorous drawing of tape back and forth on the approximal surfaces of the teeth. Large metal fillings or metallic inlays are excellent conductors of heat and cold and when inserted into deep-seated cavities are common causative factors of irritation from thermal shocks. Preparing a sound tooth for a shell or jacket crown by grinding its enamel and dentin and setting the crown with an oxyphosphate cement usually produces a very severe hyperemia which may lead to the death of the pulp. Electric shocks as a sequence of contact between an electro-positive metal, *i. e.*, an amalgam filling, and an electro-negative metal, as a gold crown, for instance, with an interposed electrolite, *i. e.*, saliva, and thus forming a completed circuit, are frequently observed. These electric shocks at times produce spasms of acute pain.

During severe damp cold weather, cases of obscure idiopathic hyperemia of the pulps of the upper and, less so, of the lower teeth are frequently observed. These acute manifestations of pain are secondary disturbances and should be classified as borderline diseases of the teeth and their adjacent structures.

Disturbances of the vasomotor system of the bloodvessels of the pulp brought about by direct or indirect irritation of these vessels is the primary cause of hyperemia.

Varieties.—Clinically, only the pathologic type, *i. e.*, obstructive (venous) hyperemia is observed. Acute hyperemia of a physiologic

type plays no part in our discussion; if it is caused by an irritant it is of very short duration and usually disappears with the removal of its cause.

Clinical Pathology.—The clinical picture of hyperemia of the pulp is portrayed by a patient complaining about a recent disagreeable sensation in a tooth, especially toward morning. Hot and principally cold fluids, or even a breath of cold air, produce a more or less sharp pain. The tooth may have been recently filled, or a cavity or some other form of structural defect of its enamel surface may be present. In the aged, senile atrophy of the alveolar process frequently exposes the roots of posterior teeth, which in consequence become highly sensitive to thermal changes.

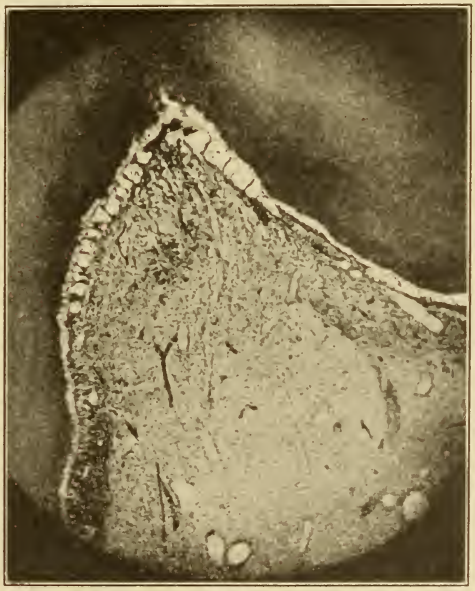


Fig. 63.—Hyperemia of dental pulp.

Pathologically, hyperemia in its chronic form is the most frequently exhibited type of pulp lesions; it is the progenitor of practically all acute or chronic diseases of this organ. The hyperemic pulp, except in traumatic cases, is always covered with a more or less sound layer of dentin. During the later stages of caries, when this layer becomes softened, infection of the pulp is practically always to be expected. Chronic irritation of the pulp leads to a renewed activity of its odontoblasts, resulting in the deposition of calcium salts. Within the sound dentin these additional deposits produce the translucent zone of Tomes, *i. e.*, a partial calcification of the dentinal fibers. When confined to the

pulp proper the deposition results in adventitious dentin along the walls of the root canal and in pulp nodules within the body of this organ. Pressure from these deposits may lead to painful acute or chronic irritation and often to complete atrophy of the pulp. Hyperemia of the pulp, histologically, manifests itself in the dilatation of all bloodvessels. As long as circulation continues the pulp will live. The anatomic structure of the pulp does not favor the establishing of collateral circulation; hence, if the circulation becomes completely obstructed stasis occurs which quickly leads to its death.

Subjective Symptoms.—On taking hot and especially cold fluids in the mouth, or from a breath of cold air, an unpleasant sensation is felt or it may manifest itself as a paroxysm of sharp pain lasting for some seconds or a minute or two only.

Diagnosis.—The patient is always able to point to the affected tooth. The antecedent history accompanied by an inspection may reveal a recent filling, a shallow cavity or some other defect of the crown, or an exposure of the root. The pulp is not exposed. The temperature test with cold water or the ethylchlorid spray is quite positive, while changes in the color of the tooth, percussion, palpation and roentgenogram are usually negative. The hyperemic pulp shows a slight increase in its reaction to the faradic current.

A *differential diagnosis* between simple irritability and pathologic hyperemia may be readily established. If a pulp which has distinctly responded to cold water resumes its normal equilibrium when the tooth is again surrounded by the physiologic fluids of the oral cavity the disturbance should be classified as simple irritability. If, on the other hand, the pain or even an unpleasant sensation lasts for some minutes thereafter, we are entitled to speak of the condition as pathologic hyperemia.

Prognosis.—Hyperemia of the pulp in its early stages usually responds favorably to conservative treatment. Under proper management resolution will occur in most cases.

Treatment.—The basic principle of the treatment of hyperemia of the pulp consists in the removal of the irritant, *i. e.*, interposing a non-conductor between the focus of irritation and the outside source. In mild forms, suitable preparation of the cavity, its sterilization with a concentrated (50 per cent) solution of thymol in aceton or alcohol, the application of a quick drying hard cavity varnish to act as an insulator, and occluding the cavity with cement either as a final filling or as an intermediate step prior to the insertion of a metal plug, is usually crowned with success. Severe cases require the application of antiseptic drugs sealed into the cavity with a temporary filling material. A gutta-percha preparation or a cement of the oxysulphate type (Fletcher's artificial dentin) or Protem cement is most convenient for this latter purpose. The selec-

tion of a suitable antiseptic is largely a matter of choice. Oil of clove, pure or camphorated liquid phenol or chloro-phenol, creosote, thymol, etc., either singly or combined answer the purpose well. All of the enumerated agents exhibit, as a side action, local anesthetic and incidentally astringent properties. Existing pain which is the sequence of an irritation will be automatically eliminated by the eradication of its cause. The medication should remain in the tooth for twenty-four hours, and, if need be, it may be renewed. If a more efficient sedative compound is desired a saturated solution of chloretone in oil of clove deserves to be recommended.

Medicaments should not be sealed into a tooth in a haphazard manner in regard to time. Usually the medicament becomes exhausted in about twenty-four hours and must be renewed if further action is to be expected. To avoid disagreeable thermal shock from sealing a medicament in a deep-seated cavity it should always be slightly warmed prior to its insertion. After the pulp has resumed its normal activity the cavity is finally sterilized, varnished and filled as described above, preferably with cement only, for a suitable length of time for observation.

Distressing cases of hyperemia of the pulp are represented by those teeth in which, through senile atrophy of the alveolar process, a large part of the root is continuously exposed to thermal changes and to irritation by foodstuffs. The palatine roots of the upper molars, and less often the roots of the lower molars, are primarily represented by this group. As these exposed roots are not amenable to protection by a filling, the only therapeutic measure which promises relief is the application of silver nitrate in a concentrated solution. The soft tissues surrounding the exposed root are protected by a coating of vaselin or a napkin, and the root itself is dried with cotton and warm air. A very concentrated solution of silver nitrate may be quickly made by crushing a few crystals on a glass slab and adding a few drops of distilled water. With a thin spatula-shaped stick of orange wood dipped into the solution, the entire area is thoroughly coated, and if possible the tooth root is exposed to direct sunlight for some minutes, or by reflecting the light into inaccessible places with the mouth mirror. In due time an intense black color is obtained. Silver nitrate may be more quickly reduced by using a 1 per cent solution of potassium or sodium hydroxid in distilled water. The treatment must be repeated two or three times on alternating days until a jet-black color of the exposed dentin is obtained. If some of the silver solution should accidentlly come in contact with the soft tissues a concentrated solution of sodium chlorid should be applied at once. Sodium chlorid is the chemical antidote of silver nitrate and changes it to an insoluble silver chlorid.

The removal of enamel from a healthy tooth by grinding, etc.,

preparatory to attaching a shell or jacket crown, in most instances produces a very painful reaction, *i. e.*, an obstructive hyperemia of its pulp. If the operator decides not to destroy the pulp it must be protected prior to setting the crown with cement against the

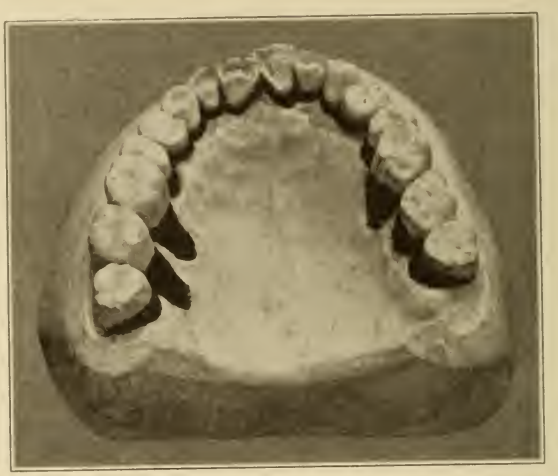


FIG. 64.—Hyperemia of the pulps of the upper teeth caused by senile atrophy of the alveolar process and the cementum and exposure of the dentin. The exposed dentin has been treated about twice a year on two to three visits with a concentrated solution of silver nitrate and has kept the patient free from pain during a period of five years.

future irritation of this vehicle, otherwise its death in due time is almost certain to occur. A heavy coating of silver nitrate until a jet-black color is obtained, as described above, and coating the black stump with a suitable hard varnish will restore the hyperemic pulp to its normal activity and protect it against future irritation.

CHAPTER IX.

ACUTE SIMPLE PULPITIS.

Synonyms.—Acute simple inflammation of the dental pulp, idiopathic pulpitis, traumatic pulpitis, *pulpitis acuta simplex, pulpitis idiopathica, pulpitis traumatica.*

Definition.—An acute partial or total exudative (destructive) inflammation of the unexposed pulp.

Etiology.—Acute inflammation of the pulp is primarily caused by bacterial invasion, usually a streptomycosis of a mixed type, arising as a sequence of existing dental caries. Numerous other primary and secondary conditions, however, which have been discussed in detail under the subject of "Etiology," play important roles as etiologic factors.

Varieties.—Clinically, only one variety is observed, *i. e.*, acute pulpitis. Depending on its etiologic nature, clinicians occasionally refer to this disease by such specific terms as idiopathic or traumatic or toxic pulpitis, etc. The disease may manifest itself as a partial or a total pulpitis; the clinical differentiation between these two forms, which are purely quantitative and *not* qualitative concepts, is too hazy to permit of a distinctive diagnosis. Partial pulpitis probably always precedes the total involvement of the pulp.

Clinical Pathology.—The clinical picture of acute simple pulpitis is usually readily recognized from a description of the manifested pain as subjectively experienced by the patient. The tooth, in most instances, shows a carious defect or some form of deep-seated abrasion and can always be pointed out by the patient; in cases of idiopathic pulpitis the correct location of the involved tooth becomes more difficult. The pulp is not exposed except in certain traumatic cases.

Pathologically, acute simple pulpitis portrays the typical picture of an acute inflammation; it is always the sequence of a neglected obstructive hyperemia and is primarily caused by a bacterial invasion. It should be borne in mind, however, that an inflammation may be produced by purely mechanical (pulp nodules), thermal (rapidly revolving disks or "setting" of certain oxyphosphate cements) or chemical (arsenic) irritation without bacterial infection, and that aseptic inflammation with aseptic pus may be the result. The routine manifestations as they gradually develop during the inflammatory process in ordinary soft tissues, as previously

discussed, must of necessity also take place in an inflamed pulp with this vital difference—that in the latter instance the cardinal symptoms of inflammation with the exception of pain cannot be or are only rarely observed in a tooth *in situ*. It should be borne in mind that a pulp may become diseased and may die without manifesting the slightest sensation. Atrophy of the pulp, which, however, is not an inflammatory process, frequently brings about such changes.

In very rare cases a pulp may die as a sequence of some general diseases, as leukemia for instance, without manifesting any pain. Sclerotic nerve degeneration as it occurs in locomotor ataxia occa-

Fig. 65.—Acute simple pulpitis.

sionally produces complete anesthesia of the dental pulp and subsequent painless inflammation and death. These disturbances are discussed under the subject of "Secondary Pulpitis."

In the early stages of acute inflammation only that part of the pulp, usually a horn, which is located opposite the point of infection, becomes involved. Consequently, the initial picture represents a partial pulpitis. Depending upon the severity of the infection, the inflammation spreads more or less rapidly, and a total pulpitis with its numerous pathologic peculiarities is the chronologic sequence of the intensely acute process, finally resulting in suppuration.

Acute partial pulpitis in very rare instances terminates in resolution; in the majority of cases suppuration and gangrene are the

logical sequences. With the involvement of the apical tissues a long chain of secondary disturbances may be expected.

Subjective Symptoms.—The pathognomonic symptoms of an acute pulpitis are the spontaneous manifestation of pain occurring especially in the evening and at night, and its increase on the application of cold. The gradation of the severity of the pain and its duration differentiates the two types of pulpitis, *i. e.*, the partial and the total variety. In the partial type the painful paroxysm may last for some minutes; the tooth is always located by the patient and no reflex disturbances are observed. The total involvement of the pulp gradually produces an increase in the painful symptoms in regard to the length of its intervals and of its duration; the location of the involved tooth becomes progressively more difficult and

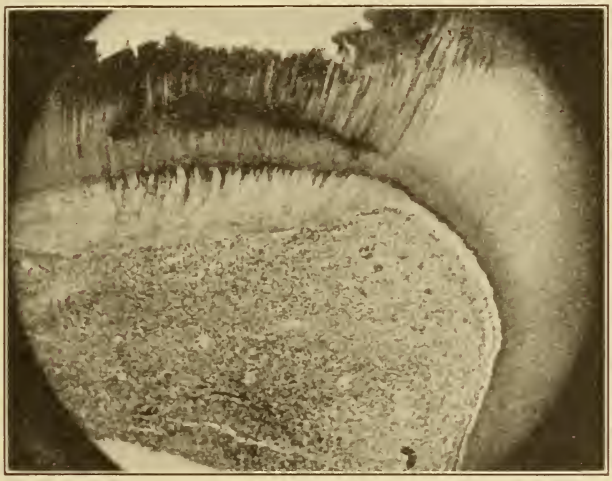


Fig. 66.—Acute simple pulpitis. A magnified section of Fig. 65 showing the early stages of inflammation of a horn of this pulp which is located opposite the point of infection.

reflex disturbances are manifested. With the increase of the inflammation there is also to be observed a gradual diminishing painful response to the application of cold, while heat becomes progressively more and more intolerable.

Diagnosis.—The painful tooth, as pointed out by the patient in the early stages of the disease, usually shows a carious defect or a traumatic lesion. On removal of the débris from the cavity, a layer of discolored and more or less decalcified dentin is observed covering a horn of the pulp The lightly passing explorer is not felt. On exerting pressure upon the decalcified pulpal wall a quick, painful response is obtained. Cold water produces a marked paroxysm of acute pain. A pledget of cotton saturated with 50 per cent alcohol and placed into the cavity produces severe painful

irritation which ceases on its removal. The color of the tooth is not altered. Percussion in the early stages of the inflammation is negative; in the last stages of total pulpitis, when the infection has passed into the periapical tissues, tapping the tooth with a steel instrument brings forth a painful response. Transillumination and roentgenogram are negative. The faradic current produces a marked sensory response from the diseased pulp which is far below the normal irritation point of the patient, indicating an acute inflammation. The more severe the inflammation, the less current is required.

The last stages of an acute total pulpitis frequently produce most violent paroxysms of pain which may radiate over a large area of the face and may last for many hours. The pain is materially exacerbated on lying down and in many respects it may simulate facial neuralgia. With the death of the pulp, the pain usually ceases unless the pericementum should have become involved.

A correct diagnosis of idiopathic pulpitis is usually fraught with great difficulties. In many instances the tooth shows no apparent extrinsic disturbances, although the true etiologic factor of this type of pulpitis, *i. e.*, the growing pulp nodule, is much favored by mechanical causes, among which advanced cases of abrasion predominate. A roentgenogram may reveal pulp nodules which, as stated, are always present, and thereby differentiate idiopathic pulpitis from hyperplasia (exostosis) of the cementum of the involved tooth. The pain, as described by the patient, is of a severe, acute, throbbing and lancinating character, and during the paroxysm, which lasts for a minute or two, the affected side of the face becomes flushed. The general character of the pain closely simulates trifacial neuralgia from which, however, it may be readily differentiated. Pathologically, idiopathic pulpitis represents a type of true neuritis of the dental pulp. It is claimed that when a tooth responds very intensely on percussion or the application of cold (ethyl chlorid) during a cycle of pain, and this hypersensation passes off immediately after the paroxysm ceases, idiopathic pulpitis is to be suspected. Its pathognomonic signs are pulp nodules; unfortunately, the roentgenogram does not always reveal their identity.

In most cases of traumatic pulpitis the pulp is exposed; rhythmic pulsation and a deep bluish-red color are observable manifestations of an acute inflammation. If the exposed pulp is pricked with a pointed instrument, a few drops of dark venous blood flow from the wound and the existing pain is much eased.

The quick response of the inflamed pulp to a low faradic current and the acute lancinating pain produced by the application of cold water diagnostically differentiates acute simple pulpitis from other types.

Prognosis—A well-established acute simple pulpitis always offers an unfavorable prognosis for the preservation of the involved pulp. The peculiar anatomic structure and other characteristics of the latter tissue, as discussed in the preceding pages, do not favor resolution. The conditions which justify an attempt to conservatively treat an inflamed pulp may be summarized by stating that only the very earliest stages of acute partial pulpitis should be selected for such procedure, provided that the particular pulp is a young growing pulp. The destruction of the pulp is always indicated in cases of total pulpitis, of severe iodiopathic pulpitis and of practically all cases of traumatic pulpitis.

Treatment.—The indications for the respective therapeutic measures depend largely upon the decision of the operator as to whether an attempt should be made to conservatively treat an inflamed pulp, or whether it should be destroyed at once. Consequently the mode of treatment selected for each procedure necessitates a separate discussion. In both instances the initial preparation is the same, *i. e.*, the tooth is placed under the rubber dam, the cavity is excavated, sterilized and a sedative antiseptic is sealed into it for twenty-four hours, and if need be, repeated. If resolution of the pulp is anticipated, the future steps are exactly the same as outlined under the "Treatment of Hyperemia of the Dental Pulp," *i. e.*, the prepared cavity is sterilized, varnished, and finally covered with cement with a view to further observation. In case the pulp is to be devitalized, the initial treatment, as referred to above (which should never be neglected), is followed by the surgical or therapeutic devitalization proper, *i. e.*, local anesthesia as a preliminary procedure is instituted or arsenic is sealed into the cavity. Both methods are discussed under the caption "Devitalization of the Dental Pulp." The removal of the pulp in a case of idiopathic pulpitis does not require any further discussion; it is precisely the same as indicated above.

CHAPTER X.

ACUTE SUPPURATIVE PULPITIS.

Synonyms.—Acute suppuration of the dental pulp, purulo-gangrenous inflammation of the dental pulp, *pulpitis acuta purulenta seu purulo-gangrænosa.*

Definition.—An acute suppurative (destructive) inflammation of the exposed pulp accompanied by the formation of abscesses or phlegmonous infiltration.

Etiology.—In the greatest majority of cases it is brought about by an infection from organisms connected with the carious process, *i. e.*, a mixed streptomycosis, although secondary infection from a chronic suppurative pericementitis or hematogenous causes are occasionally observed. Suppurative pulpitis is usually restricted to filled teeth of the permanent set, *i. e.*, teeth in which at the time of placing the filling a minute break in the continuity of the pulpal wall has been overlooked or where an infected layer of decalcified dentin covering the pulp has been inefficiently sterilized.

Varieties.—Clinically, only one variety is known. It manifests itself as an acute type, although a subacute, chronic or a purulo-gangrenous form are occasionally observed. The line of demarcation between these various grades is too hazy to be determined clinically with any degree of satisfaction.

Clinical Pathology.—The clinical picture of a case of suppurative pulpitis is always typical. The patient suffers excruciating, intolerable pain. In fact, the pain is so intense that it may be classified with the severest forms of suffering to which human flesh is heir to. The pain is not localized and is of a violent boring, throbbing character, corresponding to the rhythmical beating of the pulse. The affected tooth cannot be pointed out by the patient, and it may even be impossible for him to designate the jaw in which it is located. In the last stages of severe cases general symptoms, such as chills and fever or marked weakness due to loss of sleep, and to suffering, etc., accompany the local manifestation.

Pathologically, purulent pulpitis typifies a circumscribed suppuration of the pulp, *i. e.*, an abscess. The abscess may be single or multiple. The circulation is much obstructed, since the infected pulp has lost all power of defence; the progressive obstruction finally results in a circumscribed stasis and death of the necrobiotic portion of the pulp. The vessel walls break down; polynuclear

leukocytes, and, to a less extent, red corpuscles, are attracted toward certain bacteria in enormous quantities and thereby become the progenitors of pus corpuscles. A circumscribed collection of this freshly formed pus is designated as an abscess. In its early stages the abscess is usually confined to a horn of the pulp and is located directly opposite the point of entry of the infection, namely, the minute opening into the pulpal covering. Above the abscess a wall of inflamed tissue denotes the line of demarcation, and the remaining pulp presents the typical picture of a severe acute inflammation. With the progressive advance of necrobiosis, the final submission of the whole pulp to the infection is only a question of time. It should be observed that the process of destruction is in most cases intensely acute. The nerve fibers preserve their integrity to the very end of the struggle. As the pulp is confined

FIG. 67.—Acute suppurative pulpitis.

within an unyielding wall of dentin, the increasing pus produces compression and irritation of these fibers, and hence the persistent intolerable pain.

If the pus does not remain localized, but infiltrates the spaces between the connective-tissue cells, a phlegmonous suppuration of the entire pulp results which quickly kills it. Through the agencies of specific proteolytic ferments the necrosed tissue changes to gangrene. In multi-rooted teeth, especially the molars, this purulo-gangrenous type of pulpitis is most frequently observed. In such cases one root canal may be found to be totally gangrenous, while another canal may contain a pulp remnant in an advanced state of severe acute inflammation.

Subjective Symptoms.—Very violent, throbbing, continuous pain, increasing on entering a warm room, on lying down, or on the application of heat. Cold applied to the tooth does not seem to

affect it very much. Slowly increasing, persistent pain usually has been present for many hours or even days.

Diagnosis.—On inspection of the oral cavity, one should always be suspicious of a filled tooth of the permanent set as being the one involved. Its color is usually not altered. Transillumination may furnish a diffused shadow picture of the coronal portion of the pulp. On applying heat a marked increase of pain is felt, while cold in many instances seems to palliate temporarily. This symptom is typically pathognomonic of suppurative pulpitis. Percussion, especially in the later stages, indicates an involvement of the peridental membrane; there is no swelling of the gum tissue.

Roentgenogram is usually negative. The resistance to the electric current is materially lessened, *i. e.*, the amount of current necessary to bring about a response is much higher than that which is required to establish the normal irritation point of the patient. By carefully inspecting the pulpal wall of the excavated cavity a minute break in its continuity or an exceedingly thin elastic layer of decalcified dentin is usually observed.

Differential Diagnosis.—The intensity and duration of the throbbing pain and its increase on applying heat or by any other means which raises the blood-pressure differentiates suppurative pulpitis from other types.

Prognosis.—Resolution of a pulp affected with suppurative pulpitis is not to be expected; its removal is indicated. The subsequent treatment of the root canal, etc., usually restores the tooth to its normal functions.

Treatment.—On penetrating into a pulp chamber containing a suppurating pulp with a bur, practically no pain is experienced; occasionally, however, a very short paroxysm may be observed. On withdrawing the bur, the pent-up pus wells up, followed by a drop or two of dark blood. The violent pain which the patient has suffered for hours or days ceases almost instantly. The pulp chamber should be opened as widely as possible and washed out with tepid water. A broach may now be inserted to ascertain whether the whole or only a part of the pulp has succumbed to the infection. In the former case immediate extirpation of the necrotic pulp débris is indicated. A dressing of dichloramin-T is sealed into the empty root canal and the patient is dismissed with the general directions given below. The subsequent treatment of such root canals is discussed under "Necrosis and Gangrene." If, however, a part of the pulp is still alive, an effort is made to gently remove the necrotic portion until the line of demarcation is reached. Copious washing with tepid water will, by depletion, materially assist in quieting the intensely congested pulp stump. This antiphlogistic measure will be gratefully appreciated by the patient.

A sedative antiseptic is now sealed into the dried pulp chamber

for from twenty-four to forty-eight hours. Suitable compounds for such purposes consist of a saturated solution of chloretone in oil of clove or a 20 per cent solution of novocain in camphorated phenol. No effort should be made at this time to extirpate the remaining pulp stump under pressure anesthesia. Violent pain will always follow an attempt of anesthetizing an inflamed tissue by direct contact. Even conduction anesthesia in such cases is not always followed by its customary success. To apply arsenic upon an inflamed pulp indicates utter disregard of the fundamental principles of pharmaco-therapy; intolerable pain is always the sequence of such a procedure. On return of the patient the now quiescent pulp stump will usually allow its extirpation under some form of local anesthesia or arsenic application as discussed under "Devitalization of the Pulp."

The general condition of a patient suffering with suppurative pulpitis demands careful attention. Light nutritious food is advised, and the patient instructed to go to bed as soon as possible in order to refresh himself with a long and much needed sleep. Before retiring, as a prophylactic, 10 grains (0.6 gm.) of acetylsalicylic acid (aspirin) are taken with a glass of water or a cup of warm tea. A saline purge given the following morning is in most cases indicated.

CHAPTER XI.

SECONDARY PULPITIS.

Synonyms.—Secondary inflammation of the dental pulp, *pulpitis secundaria*.

Definition.—A secondary inflammation of the unexposed pulp as a sequence of a local or a general disease.

Etiology.—Secondary pulpitis may be caused by a primary existing local disturbance within the region of the affected tooth or by a general disease. In the majority of cases the causative agent is the sequence of a local or general infection, although general diseases of a non-infectious type, such as leukemia, locomotor ataxia, chlorosis, increased intradental blood-pressure and various neuropathic conditions, as hysteria, neurasthenia, etc., may be the exciting factors.

Varieties.—Clinically, secondary pulpitis may manifest itself as any one of the numerous varieties of pulpitis, *i. e.*, its course may run from an obstructive hyperemia to necrosis or gangrene of the pulp, although usually it is restricted to the hyperemic type.

Clinical Pathology.—The clinical picture of secondary pulpitis depends primarily upon the underlying disease and as a consequence it varies accordingly very widely indeed. If the primary disturbance of the pulp is of a purely local character the resulting secondary pulpitis may present itself as any one of the numerous modifications as outlined above and its early recognition should offer no difficulties. If, on the other hand, the underlying cause is a general disease the clinical picture becomes much diffused. The patient complains of pain, which he usually designates as being of a continuous, dull character. He is not able to locate the respective tooth, but merely points to the affected side of the face. Such disturbances usually manifest themselves as the early stages of obstructive hyperemia and they are recognized from a description of the existing pain. The teeth in most instances will be found to be intact. In certain rare cases necrosis of the pulp may exist, as in leukemia, increased intradental blood-pressure, etc., which at times, through a crack in the enamel and dentin, becomes secondarily infected and gangrene with its numerous consequences results. These latter disturbances are also readily diagnosed. In rare instances, as in locomotor ataxia, the secondary pulpitis manifests itself by a total absence of sensation due to nerve degeneration or as a degen-

eration of the entire pulp, and it may be discovered by accidentally or intentionally entering into the pulp chamber.

Pathologically, secondary pulpitis manifests itself within the pulp by the same symptoms as they are observed in other tissues. If the underlying cause is an infection from a local source it is usually of a mixed type and as a consequence its pathology differs nowise from that discussed respectively under the primary disease of the dental pulp. If the secondary disturbance is dependent upon a general disease a discussion of the pathology of the latter, with the exception of the general facts, must be omitted at this moment. For a further elucidation of the subject the reader is referred to works on general and special pathology.

Diagnosis.—Secondary pulpitis as a sequence of a primary local disturbance offers no difficulties in its recognition, and may be readily diagnosed by the various methods as outlined in the previous chapters. If the pulpitis is caused by a general disease an effort should be made to locate the predisposing disturbances. It is not to be assumed that the dental practitioner should boast as an expert diagnostitian of general diseases. Consequently the following diagnostic hints are merely cited as aids to a possible recognition of the primary disease, but by no means as an indication for the treatment of the respective ailment. The patient should at once be intrusted to the care of a general practitioner or a specialist for further observation.

Of the general diseases which are prone to cause secondary idiopathic disturbances in the pulps of the upper teeth preëminently acute catarrh of the maxillary sinus should be mentioned. It is of very common occurrence, indeed, and, with Arkœvy,[1] who has depicted the first clear clinical picture of the symptomatology of this disease, the writer is fully in accord that: One should not overestimate unimportant things nor neglect things which may be of great value. The ready recognition of the underlying antrum disturbances as the true cause of the existing dental ailment combined with the subsequent treatment which, incidently, is usually successful, is a source of satisfaction to both patient and operator. Hence, a detailed discussion of the clinical phases of this dental borderline disease may not be amiss.

Acute nasal catarrh, "cold in the head," is an acute catarrhal inflammation of the Schneiderian membrane, which lines the nose and its accessory cavities. It is characterized by feverishness, feeling of discomfort in the head with a free discharge of watery, mucous or mucopurulent fluid. Acute nasal catarrh is principally brought about by an infection through sudden atmospheric changes, exposure of the face and neck or the feet and ankles to cold draught

[1] Arkœvy: Oesterreich-ungar. Vierteljahresschrift, 1897, p. 40.

and dampness are among the usual causes. Automobile rides in chilly moist air are most frequent sources. By continuity of the mucous lining of the nose an acute rhinitis or any other infection of a general character, such as influenza, pneumonia, scarlet fever, tuberculosis, measles, etc., rapidly spreads to the antral sinus. Anatomically, the maxillary sinus presents in its lower floor little

FIG. 68.—Longitudinal section of a premolar showing the diseased pulp, caused by influenza. (Fischer.)

hillocks which are the apical ends of the roots of certain upper teeth, *i. e.*, principally the molars and premolars. Frequently, these root ends protrude through the lower floor and are only covered by the mucous membrane of the antral cavity. It is readily observed that an inflammation of this membrane which practically is synchronous in its blood and nerve supply with that of the adjoining teeth will rapidly spread to the pulps of the teeth within

the affected region. A dull painful sensation about the teeth and the bony region of the afflicted side of the face is the result. The patient cannot locate the painful teeth. On pressure upon the external wall of the antral sinus within the region of the canine fossa distinct pain is felt which may simulate a neuralgic paroxysm. The concomitant appearance of these symptoms points to a borderline disease of the teeth and the antrum and it should be diagnosed as an acute catarrh of the maxillary sinus and secondary obstructive hyperemia of the pulps of the involved teeth. The disease occurs principally in heavy winter weather and is much aggravated by a simultaneously existing influenza. During the winter of 1917-1918 the writer observed among the 650 students of the Evans Institute some 35 cases of this type, *i. e.*, about 5 per cent of the total number. The prognosis is always favorable.

If by accident or otherwise a pulp is entered into and it is found to be free from sensation a differential diagnosis will clarify the situation in regard to its causative factors. Senile types of degeneration (atrophy), locomotor ataxia or leukemia are the three primary diseases which may be responsible for this condition.

Degeneration of the dental pulp is of a very common occurrence, especially in abrated and in senile teeth and it is readily diagnosed in the exposed organ. (See Degeneration of the Dental Pulp.)

Locomotor ataxia is a disease principally observed in males of middle life. It originates in a chronic degeneration of the posterior columns of the spinal cord and the posterior nerve roots and is characterized by loss of coördination, neuralgic pains in the limbs, loss of sensation and trophic changes. The diagnosis is made positive by three pathognomonic signs: (1) Westphall's sign, *i. e.*, absence of the patellar reflex; (2) Romberg's sign, *i. e.*, swaying of the body and inability to maintain erect position with closed eyes; and (3) Argyll-Robertson's sign, *i. e.*, loss of pupillary reflex to light. The prognosis is always unfavorable.

The chronic degeneration of the nerve endings as an early symptom of locomotor ataxia is most interestingly demonstrated in the dental pulp and the peridental membrane. On entering into an apparently normal pulp of a patient suffering with this disease it will be observed that this ordinarily most sensitive organ will not respond to the advancing dental broach and it may be removed without the slightest manifestation of pain. Spontaneous painless loosening of sound teeth, principally of the molars and premolars of the upper jaw to an extent that they may be readily removed with the fingers, should always be looked upon as a pathognomonic symptom of an existing locomotor ataxia. Cases of this type should arouse grave suspicion and the patient should at once be put through the above-mentioned diagnostic tests. If they are positive he should immediately be referred to a physician.

Leukemia, usually an acute disease of the blood, manifests itself in an enormous increase in the number of white blood corpuscles, with enlargement of the lymph nodes, the spleen, etc. In the mouth leukemia produces characteristic symptoms which are readily recognized by the trained observer. The gums are swollen, easily bleeding on being wounded, especially near the dental papillæ, and the palate is often covered with petechial spots of coagulated blood. The gingival tissue frequently exhibits necrotic areas and presents a dirty, brownish puffed-up ridge which loosely encircles the teeth. The secretion of saliva and mucus is much accelerated and foul breath is very pronounced. In other words, the picture presents a typical leukemic stomatitis. An examination of the blood must be insisted upon to verify the diagnosis of this comparatively rare disease. The prognosis is always unfavorable.

Malaria, a paroxysmal fever, is characterized by a regular succession of definite stages, *i. e.*, cold, hot and sweating, and followed by a period of complete intermission. It is caused by a protozoön present in the blood, the *Plasmodium malariæ*. When the characteristic chills, fever and sweat occur periodically the diagnosis is practically certain, although a blood examination is often desirable. Flagg, Ware, Garretson and numerous other writers have called attention to the relationship of odontalgia (secondary pulpitis) to malarial fever. Especially significant in this respect are the observations of Marshall.[1] He relates that: "It was my fortune or my misfortune, I might say, to practice for many years in a malarial district, and I saw many cases of neuralgia arising from malarial influences, as proved by quinin in curing them without operation. I found pulpitis more difficult to control by the ordinary remedies in this district than I have found in any other place where I have practised. There seems to be a hypersensitiveness to nerve tissue in these cases, and the ordinary remedies for controlling pulpitis do not seem to have the same effect. Abscessed teeth do not respond to treatment as readily in such districts as upon high land where malaria is not present." The writer, from his own observations, is in full accord with Marshall's statement regarding the relationship between malaria and secondary pulpitis. In sections of the country infested with malaria one should always be suspicious of a possible linking-up between malarial fever and secondary pulpitis.

Increased intradental blood-pressure constitutes a most interesting etiologic factor in the production of secondary pulpitis. The credit of having first called attention in print to this interesting phenomenon belongs to Ferdinand Tanzer;[2] although the late Dr. Garretson always emphasized this very same fact in his lectures.[3]

[1] Dental Cosmos, 1892, p. 569.

[2] Oesterreich-ungar. Vierteljahrsschrift, 1905, p. 477.

[3] The writer is indebted to the late Dr. M. N. Cryer for this information.

The overfilled bloodvessels exert pressure upon their walls and wherever they meet with resistance compression results, which manifests itself as pain. From an anatomic viewpoint, it is readily understood why this manifestation of pain is so much more pronounced in the dental pulp than in other soft tissues. As a consequence, any cause which may produce a general increased blood-pressure will leave its imprint upon this delicate organ. If the respective dental pulp is pathologically altered by an overgrowth or some other developmental defect of its surrounding hard wall of dentin, or if it has undergone degenerative changes, the pain arising from a plethoric circulation, *i. e.*, a congestive hyperemia, is naturally more pronouncedly manifested in this particular diseased pulp than in a normal pulp. While any dental pulp by virtue of an existing pathologic lesion may be selected as the point of minor resistance, usually the pulps of the upper second incisors and those of all the lower incisors are more often the victims, as they may be classified as "weak" teeth. Clinical observations substantiate this conception. The resultant hyperemia manifests itself primarily as a dull, continuous pain which is much accelerated by sudden temperature changes. Aside from numerous general diseases which concomitantly produce high blood-pressure, there are specifically two physiologic factors, *i. e.*, pregnancy and menstruation, which are prone to manifest themselves secondarily as pulpitis and, as a consequence, may induce the patient to seek the services of the dental practitioner.

Secondary pulpitis as an affliction of the pregnant woman is exceedingly common either as an aggravation of some ordinary complaint caused by the mechanical effects of the uterine enlargement or it may result from a reflex neurosis due to the extraordinary stimulus acting on the genital tract, or from that interesting and little understood interference with general metabolism, *i. e.*, toxemia of pregnancy.

Disturbances of menstruation are most frequently met with in girls and young women. Practically every civilized woman suffers more or less discomfort and malaise at this period, the most common manifestations are pain and weight in the back and loins, abdominal cramps, headache, general lassitude, etc., occurring especially the days before and during the menstruation. Mild types of gingivitis occurring during menstruation and pregnancy are exceedingly common. The increased blood-pressure very frequently sets up a painful secondary pulpitis and hence the appeal to the dental practitioner.

Regarding the diagnosis of these cases, it may be stated that a carefully worded inquiry will usually reveal the underlying cause. Overscrupulous and sensitive patients require tactful handling. If the true cause is ascertained an assurance may be given that

with the termination of the physiologic disturbance the painful tooth will return to its normal state of health without further treatment. In rare instances a pulp may die from the effects of increased intradental blood-pressure.

Chlorosis, a pronounced type of anemia, is principally met with in young girls about the age of puberty and it manifests itself as a marked decrease of the hemoglobin content of the blood. It is chiefly associated with disorders of menstruation. As a not infrequent complication, a dull, continuous toothache in otherwise apparently sound teeth is observed. The underlying cause of this type of secondary pulpitis is usually recognized at once by the color of the patient. The change of the complexion is marked, viz., blonds become pallid and waxy and brunettes muddy and grayish with bluish-black rings under the eyes. Chlorotic patients often manifest an increased tendency to dental caries. At present it is assumed that chlorosis causes a disturbance of internal secretions and the resultant change interferes with the calcium metabolism and thereby creates a predisposition to dental caries. Suitable treatment consisting of plenty of food, fresh air, rest and iron will assist the patient in her return to normal health and thereby incidentally the secondary pulpitis is eradicated.

Prognosis.—The prognosis of a pulp afflicted with secondary pulpitis depends upon its original cause. If the secondary disturbance is brought about by a local infection of the investing tissues of the tooth, the concomitant routine treatment of the initial cause and the existing pulpitis will restore respectively the pulp or the pulpless tooth to its former usefulness. In case the secondary pulpitis is the sequence of a hematogenous infection, the prognosis for the ultimate recovery of the pulp is usually favorable. Leukemia and locomotor ataxia are unfavorable predisposing causes; these diseases usually are fatal.

Treatment.—The treatment of a pulp afflicted with secondary pulpitis depends primarily upon the recognition of its predisposing cause. If the existing pulpitis is the sequence of a local infection, a correct diagnosis of the respective condition of the diseased pulp will clearly indicate the mode of its treatment as outlined in the previous sections. It is understood that the primary cause, of necessity, must also be eradicated. If the secondary pulpitis manifests itself as an obstructive hyperemia, or even a mild type of acute pulpitis as a sequence of a hematogenous infection caused by a general disease, such as an acute catarrh of the antrum, influenza, malaria, etc., or an exanthematous disease, *i. e.*, measles, scarlet fever, etc., the successful termination of the causative disease usually restores the affected pulp to its former usefulness. The restoration of the afflicted pulp to its normal activity under these conditions furnishes a splendid example of the ancient medical

aphorism: *Cæsante causa, cæsat morbus, i. e.*, Let the cause be removed and the disease ceases. If the operator has satisfied himself that his patient suffers from a secondary pulpitis with no perceptible manifestations of local lesions and with no indications for specific dental treatment, he should always direct him to consult a general practitioner or a specialist, as the case may be, accompanied by such information as to guide him in regard to the apparent nature of the underlying ailment.

The treatment of a type of secondary pulpitis which, as we have pointed out above, should be classified as a dental borderline disease, *i. e.*, the simultaneous appearance of an acute catarrh of the maxillary sinus and an obstructive hyperemia of the pulps of the upper teeth of the afflicted side, requires detailed discussion. A patient suffering with this most annoying acute disturbance, which manifests itself as a severe continuous, dull, painful sensation of the affected side of the face and principally of the teeth within this region, usually consults his dentist and insists—and rightly so—that the pain is primarily located about his teeth. If the dentist has correctly diagnosed the condition the following symptomatic treatment will give excellent results. The patient is ordered to bed, light nutritious diet is prescribed and he is advised to take 10 grains (0.65 gm.) of acetyl-salicylic acid (aspirin) every two hours with a cup of hot tea or a glass of water. Before going to sleep a hot lemonade is advised to promote free perspiration. Twice during the day (mornings and evenings) the afflicted side of the face should be placed upon the hot-water bag for a half hour. The nose and the mouth should be rinsed at frequent intervals with a warm physiologic saline solution. The patient should be cautioned against exposure to draughts as acetyl-salicylic acid seemingly predisposes to aërobic infection. Usually within a few days all symptoms have disappeared and complete recovery is assured.

Occasionally a somewhat similar effect of an exposure to chilly damp air manifests itself in the lower jaw. A painful obstructive hyperemia of the pulps of the lower teeth of the affected side is the direct sequence. Principally, however, it is the temporo-maxillary joint which has to bear the brunt of the burden and it responds with a painful, acute inflammation of its synovial membrane. On opening of the mouth a "cracking" sound or a distinct momentary locking is observed, which is the result of the swollen intra-articular fibrocartilage being caught by the moving condyle. Pressure with the finger in front of the tragus of the ear and with the mouth wide open will cause a most painful sensation within that region which verifies the diagnosis. The condition may be referred to as an acute mandibular arthritic inflammation or as an arthro-rheumatic affection of the temporo-maxillary joint, *i. e.*, rheumarthritis.

The treatment of this dental borderline disturbance, *i. e.*, secondary pulpitis combined with inflammation of the mandibular joint, is precisely the same as that suggested above with one important adjunct, *i. e.*, temporary rest of the joint. The simplest method to obtain this required rest consists in applying a head-and-chin bandage padded thickly with cotton about the affected region to be worn during the night and if possible the greater part of the day. Salicylic acid in the form of an ointment, either as the Compound Menthol Ointment, N.F., or in the form of the ready-made Baume Analgésique Bengué, should be thoroughly rubbed into the skin about the affected region on retiring. In addition, 10 grains (0.65 gm.) of atophan, combined with 10 grains (0.65 gm.) of sodium bicarbonate and suspended in a large glass of cool water, should be taken twice a day between meals.

CHAPTER XII.

CHRONIC ULCERATIVE PULPITIS.

Synonyms.—Chronic ulcerative or parenchymatous inflammation of the dental pulp, *pulpitis chronica ulcerosa seu parenchymatosa*.

Definition.—A chronic ulcerative inflammation of the exposed pulp.

Etiology.—The causes of chronic ulcerative pulpitis are practically always to be sought for in a mixed streptococcus invasion arising from the carious process through a more or less extensive break in the pulpal wall.

Varieties.—Only one variety is known.

Clinical Pathology.—The clinical picture of this disease is somewhat hazy; only mild and frequently no subjective manifestations are observed. The affected tooth usually has a deep cavity or a badly leaking filling extending into the pulp chamber which allow free drainage of the products of suppuration. Occasionally a patient complains of a disagreeable sensation in the tooth, although real pain is rarely felt except when pressure from food débris crowded into the cavity produces a paroxysm which may last for some minutes, or until the pressure is relieved. No swelling of the surrounding tissues is observed.

Pathologically, the exposed pulp presents a moist grayish-white necrobiotic surface, *i. e.*, a suppurating ulcer. Below the débris a wall of dense granulation tissue is felt with a slowly advancing explorer which separates the necrotic tissue from the underlying chronically inflamed pulp. In the struggle for existence against the constant irritation brought about simultaneously by the infection and the pressure from decomposing food crowded into the open pulp chamber, the pulp responds gallantly with all its forces of defense which results in the above referred to line of demarcation and leads to the deposition of adventitious dentin and pulp nodules within its own tissue. These nodules grow concentrically and may assume a size so large as to completely fill the pulp chamber or the root canal. The further disintegration of the remaining pulp is a very slow process. Depending upon the original vitality of the pulp, it may require many months for its completion.

Subjective Symptoms.—The patient complains of very little discomfort; when food is crowded into the cavity, pain from pressure results which is relieved by its removal. Only in very advanced cases are secondary manifestations observed.

Diagnosis.—Chronic ulcerative pulpitis may be accidentally discovered by an inspection of the teeth. On excavating the cavity a break in the pulpal wall is revealed; the pulp chamber is filled with necrobiotic tissue, and on inserting an explorer into the inflamed portion of the pulp severe pain is manifested. The application of cold produces pain, indicating inflammation; percussion is negative. The roentgenogram may show pulp nodules. The response to the electric current is usually the same as that observed in suppurative pulpitis, namely, a greater amount of current is necessary than normally. Chronic ulcerative pulpitis is a comparatively rare disease which at times offers great difficulties in its recognition; usually it is only observed in the neglected teeth of adults.

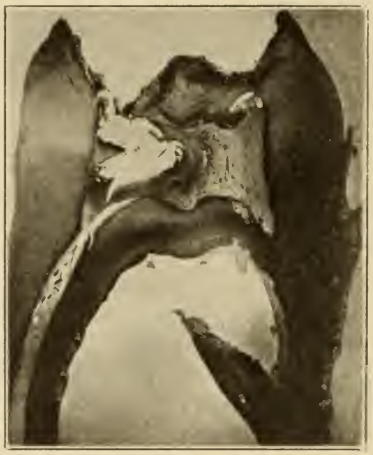


Fig. 69.—Chronic ulcerative pulpitis.

Differential Diagnosis.—An open pulp chamber and comparative freedom from pain are the principal pathognomonic signs of chronic ulcerative pulpitis and differentiate this disease from other types.

Prognosis.—A chronically inflamed pulp with an ulcerating surface does not offer favorable chances for its preservation; the removal of the diseased pulp and subsequent treatment of the root canal restores the tooth to normal function.

Treatment.—Chronic ulcerative pulpitis calls for the same routine procedures as previously described under "Acute Suppurative Pulpitis."

CHAPTER XIII.

CHRONIC HYPERPLASTIC PULPITIS.

Synonyms.—Chronic hypertrophy of the dental pulp, pulp polypus, productive inflammation of the dental pulp, *pulpitis chronica hypertrophica, seu granulomatosa, seu plastica.*

Definition.—Chronic hyperplastic pulpitis is a chronic productive inflammation of the exposed pulp characterized by a slowly growing granulation tissue upon its surface, filling more or less the carious cavity and sometimes protruding therefrom. Correctly speaking, a polypus should be designated as a pedunculated swelling or outgrowth of a mucous membrane and hence the term pulp polypus is misapplied.

Etiology.—It is caused by a chronic irritation of a young growing pulp due to the rough edges of the broken walls of the pulp chamber. A large opening into the chamber seems to be imperative. The fungoid growth may be looked upon as an endeavor instituted by the irritated pulp to protect itself.

Varieties.—One variety only is observed; it may occur as a simple small overgrowth of the irritated pulp tissue, or as a vascular mass filling the entire carious cavity or even protruding therefrom. On rare occasions the granulation tissue may relatively assume an enormous size.

Clinical Pathology.—The clinical picture of a chronic hyperplastic pulpitis, commonly referred to as pulp polypus, is very typical. Within the cavity of the tooth a deep red mass of granulation tissue, ranging in size from that of a pinhead to a large pea and often protruding from the cavity may be seen. In fractured teeth a fungoid growth of the exposed pulp is occasionally observed. On touching the growth with the finger, pulsation may be felt; the patient does not complain of real pain. A slight wound in its surface causes a profuse hemorrhage. Pathologically, this new growth is a typical hypertrophy and consists of slowly growing granulation tissue endowed with a rich blood supply and an absence of nerve fibers. Correctly speaking, it is *not* a neoplasm (tumor). The odontoblasts disappear, hence the polypus does not respond painfully on being touched except upon increasing pressure, which calls forth a reaction from the pulp proper. With the increase in its size the polypus may protrude from the cavity of the tooth, and by its close contact with the gum tissue may become covered

with epithelium, as a consequence of mechanical irritation. It should be remembered that the normal pulp does not contain this latter tissue, hence the observed epithelial covering is the result of a process of autotransplantation from the surrounding mucous membrane.

Chronic hyperplastic pulpitis is a comparatively rare disease; it is usually restricted to molar teeth with large crown cavities, and is most frequently observed in the neglected oral cavities of children. It may be looked upon as an expression of a powerful defensive process brought about by chronic irritation of a young growing pulp present in the mouth of an otherwise healthy individual.

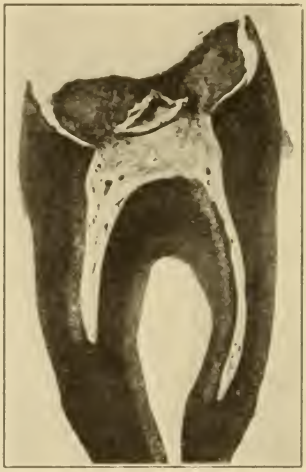


FIG. 70.—Chronic hyperplastic pulpitis. Pulp polypus.

Subjective Symptoms.—Subjective symptoms manifest themselves as an unpleasant sensation, which, however, is not expressed as real pain. Upon pressure from food crowded into the cavity or from other sources of irritation a painful response is obtained from the chronically inflamed pulp. Secondary symptoms are rarely observed.

Diagnosis.—Chronic hyperplastic pulpitis is readily diagnosed by physical inspection; its principal diagnostic features are comparative freedom from pain on slight pressure, very free bleeding when wounded and the presence of one or more pedunclcs which attach the granulation tissue to one or more horns of the pulp. An explorer inserted into the pulp proper calls forth a very painful reaction, as the pulp is always found in a chronic state of mild inflammation. The electric current verifies this latter diagnosis. Pericementitis as a sequel is rarely observed.

The *differential diagnosis* is primarily concerned with the differ-

entiation between the pulp polypus and the gum tissue which may have grown into the cavity. The rough edges of the cavity may irritate the gum tissues and ingrowing hypertrophic gum is a very common occurrence. The latter also bleeds very freely, but is slightly more painful on being injured. Lifting up the questionable tissue usually reveals its identity. In cases of doubt packing the cavity with temporary stopping for twenty-four hours will clarify the diagnosis.

Prognosis.—As the pulp is always found in a more or less pronounced state of chronic inflammation, its destruction and removal is indicated. The prognosis of saving the pulpless tooth usually is good.

Treatment.—The treatment of chronic hyperplastic pulpitis conrists in the preliminary surgical removal of the granulation tissue and destruction of the remaining pulp. To facilitate the painless amputation of the polypus a drop of liquid phenol is allowed to flow upon it and very shortly the cauterized head, which now appears as a milk-white bead, may be cut off with a sharp spoon curette or a hatchet-shaped excavator, or with a small curved lancet. Profuse bleeding results. The further treatment, *i. e.*, the removal of the pulp, etc., is carried out as discussed under "Devitalization of the Dental pulp."

CHAPTER XIV.

DEGENERATION OF THE DENTAL PULP.

Synonyms.—Chronic parenchymatous pulpitis; atrophy of the pulp, *pulpitis chronica parenchymatosa*, *atrophia pulpæ*.

Definition.—Chronic parenchymatous degeneration of the pulp.

Etiology.—Atrophy of the pulp is primarily caused by natural senile, retrogressive, metamorphotic changes within the pulp tissue independently of inflammatory processes. Pathologists recognize amyloid, atrophic, fatty, fibroid and calcareous varieties.

Clinical Pathology.—In the absence of any recognizable symptoms of the unexposed pulp there is no clinical picture to be observed.

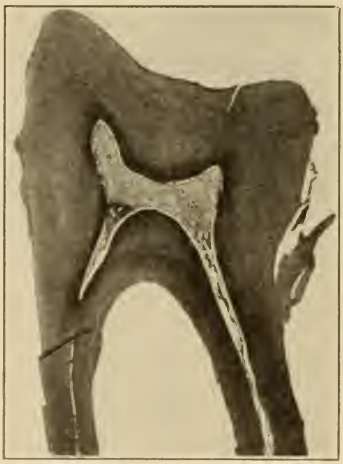


Fig. 71.—Degeneration of the dental pulp.

Pathologically, degeneration of the pulp is most frequently and, incidentally, rather commonly met with in the teeth of the aged; it is an eminently chronic process. It may be restricted to the periphery of the pulp; usually, however, it involves the whole organ. The degenerated pulp presents the typical picture of a retrogressive metamorphosis, *i. e.*, dilated bloodvessels, degenerated nerve fibers, reticular atrophy or adenoid degeneration of its connective tissue and reduction of the size of the odontoblasts. The various types of degeneration present definite pathologic phases, which are only recognizable microscopically. As the clin-

ical practitioner cannot diagnose atrophy of the pulp *in situ*, an *ex cathedra* discussion of its pathology would prove of little service to him. For further information on this subject the reader is referred to the works of Hopewell-Smith, Black, Wedl, Walkhoff, Roemer, Bennet, etc.

Subjective symptoms are absent.

Diagnosis.—An unexposed atrophic pulp cannot be diagnosed. More or less complete absence of sensation with negative findings in regard to the color of the tooth, to conductivity of temperature, transillumination, percussion and the roentgenogram furnish sufficient proof to substantiate the above dogmatic statement. The

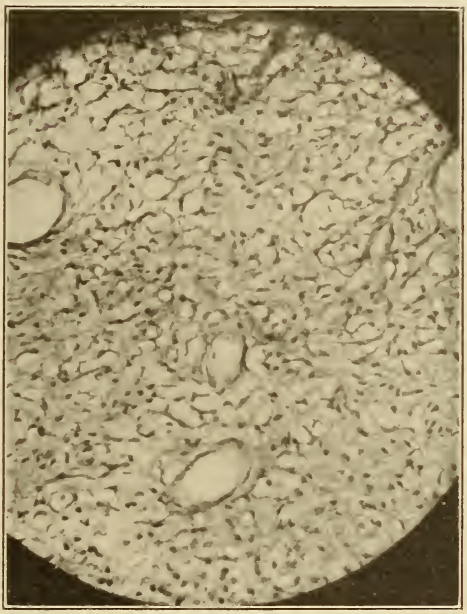


FIG. 72.—Degeneration of the dental pulp. Magnified section of Fig. 71.

slightly lowered resistance of a pulp undergoing atrophic changes to the faradic current and other diagnostic aids are too meager to be of assistance in the recognition of degenerative changes occurring within this organ. Naturally, a completely degenerated pulp is a "dead" pulp, and, as a consequence, no response is obtained by the electric current.

Prognosis.—The extirpation of the atrophic pulp and the subsequent routine treatment and filling of the root canal restores the affected tooth to its former usefulness.

Treatment.—On opening the pulp chamber of a tooth by intent or accidentally containing an atrophic pulp, the operator at once is

forcibly impressed with three jointly existing conditions which are contrary to his expectations, *i. e.*, a partial or more often a total absence of all sensation, no hemorrhage and an absolute freedom from putrescent odor. The extirpated pulp presents itself as a shriveled wax-like or fibrous thread of a yellowish transparent color or having a reddish-brown tint. On crushing the extirpated tissue, pulp nodules may be felt. The clinical diagnosis of degeneration of the dental pulp is assured by these pathognomonic findings. Most frequently the root canals will be found partially or totally obliterated by calcareous deposits. The root canals, emptied of its contents, in the absence of a bacteriologic examination, should always be looked upon as infected and treated accordingly as outlined under "Necrosis and Gangrene."

CHAPTER XV.

DEVITALIZATION OF THE DENTAL PULP.

The successful application of therapeutic measures to a diseased pulp for the purpose of bringing about its resolution unfortunately is restricted to very limited bounds. The only absolute safe prognosis of an inflamed pulp consists in its destruction. Clinical experience bears out the fact that the latter procedure, if carried out under aseptic precautions, is eminently successful and the removal of the pulp does not materially jeopardize the future usefulness of the involved tooth. Aside from numerous pathologic interferences with the normal equilibrium of the dental pulp, the practitioner is frequently called upon to destroy this organ for many other reasons.

The indications for the devitalization and removal of the dental pulp may be summarized as follows:

1. All cases which, in accordance with the judgment of the operator, offer only questionable chance for resolution, *i. e.*, exposure of the pulp, hyperemia and the early states of acute pulpitis.
2. All the other disturbances of the pulp as discussed under their respective headings in the previous chapters.
3. All cases of extensive carious destruction of the crown of a tooth involving its pulp which call for an artificial substitute.
4. Certain cases of malformation, malposition or other esthetic defects which call for an artificial substitute of the distorted crown.
5. Certain cases in which a sound tooth is to be utilized as a post for anchoring an artificial substitute.

The various methods employed for the devitalization of the dental pulp may be carried out according to the following procedures.

1. Destroying the living pulp by purely mechanical means, *i. e.*, "knocking out" of the pulp without an anesthetic.
2. Destroying the pulp under a general or a local anesthetic.
3. Destroying the pulp by the actual cautery or a potential caustic.

Destruction of the Pulp by Mechanical Means Without an Anesthetic.—In bygone days the removal of a pulp by purely mechanical means, *i. e.*, the extirpation of the exposed pulp by inserting a broach or by "knocking out," as this barbaric process is known, was in common vogue. Fortunately, it is rarely practised at present. The "knocking out" process consisted in driving a suit-

able shaped wooden stick into the pulp chamber by the quick blow of a mallet. In defense of the advocates of this method, it must be stated that the pain experienced from this procedure when dextrously performed is comparatively little, provided that the pulp is in a perfect state of health, which, however, is rarely the case and which, incidentally, cannot always be ascertained with any degree of absolute satisfaction. However, with the present methods of inducing local anesthesia there is no valid reason for the continuation of such heroic procedures.

Destruction of the Pulp Under a General or a Local Anesthetic.—As a general anesthetic produces insensibility of the pulp precisely in the same manner as in any other organ of the body, no further discussion is necessary at this moment. The administration of nitrous oxid or some other general anesthetic agent is occasionally employed for such purposes. The so-called "dental analgesia" is *not* to be advised for the "painless" removal of the pulp. The administration of a local anesthetic for this specific purpose is at present so eminently successfully and widely practised, that its technic, of necessity, must be discussed in detail.

Injection into the Pulp (Pressure Anesthesia).—By pressure anesthesia, pressure cataphoresis, pulp anesthesia or contact anesthesia as this process is variously termed, we understand the introduction of an anesthetizing agent in solution by mechanical means through the dentin or directly into the exposed pulp for the purpose of rendering this latter organ insensible to pain. Simple hand pressure with the finger or with a suitably shaped instrument, with the hypodermic syringe or with the so-called high-pressure syringe is recommended for such purposes.

Before describing the *modus operandi* of the various methods, the histologic structure of the dentin should be briefly recalled. Dentin is made up of about 72 per cent inorganic salts, about 10 percent water and an organic matrix constituting the remainder. The dentin is traversed by a very large number of tubules, radiating from the pulp cavity in a more or less wave-like manner toward the periphery where they branch off, forming a deltoid network. These tubules are filled with the processes of the odontoblasts, known at present as Tomes' fibers, and they are concerned with the metabolic changes occurring in the dentin. The dentinal fibrils are protoplasmic in their nature and normally do not carry physiologic sensation in the sense as we understand this term. When the fibers have become highly irritated a mere touch upon the dentin may at once call forth a paroxysm of pain.

1. **Devitalization of the Pulp when Exposed or Covered with a Layer of Decalcified Dentin.**—Isolate the tooth with the rubber dam, and clean it with an antiseptic solution. Excavate the cavity as much as possible, and if the pulp is not fully exposed wipe out the cavity with chloroform to remove fatty deposits from the cartilagin-

ous layer of dentin, and dehydrate with absolute alcohol and warm air. Saturate a small pledget of cotton with a warm concentrated novocain (procain) solution in sterile water, place it into the prepared cavity or over the exposure and cover it with a large pledget of cotton, and then, with a piece of slightly warmed unvulcanized rubber so as to render it plastic and which should about completely fill the cavity, and with a broad-faced amalgam plugger or some other suitably shaped instrument, apply slowly increasing continuous pressure from one to three minutes. The pulp may now be fully exposed and tested. If it is still sensitive repeat the process. Loeffler states: "This pressure may be applied by taking a short piece of orange wood, fit it into the cavity as prepared, and direct the patient to bite down upon this with increasing force. In this way we can obtain a well-directed regulated force of pressure and with less discomfort to the patient and the operator."

Miller described his method as follows: "After excavating the cavity as far as convenient and smoothing the borders of it, take an impression in modelling compound, endeavoring to get the margins of the cavity fairly well brought out; put a few threads of cotton into the cavity and saturate them thoroughly with a 5 to 10 per cent solution of novocain, cover this with a small bit of rubber dam, and then press the compound impression upon it. We obtain thereby a perfect closure of the margin, so that the liquid cannot escape, and one can then exert pressure with the thumb sufficient to press the solution into the dentin." Instead of the novocain solution, a so-called novocain "pluglet" may be used. A pluglet is introduced into the cavity, covered with a wisp of cotton dipped in sterile water and the further procedure is precisely the same as described above.

2. **Devitalization of the Pulp when Covered with a Thick Layer of Healthy Dentin.**—With a very small bi-bevelled drill bore through the enamel or directly into the exposed dentin at a convenient place, guiding the drill in the direction of the pulp chamber. Blow out the chips, dehydrate with alcohol and warm air and apply the hypodermic or high-pressure syringe, provided with a special needle, making as nearly as possible a water-tight joint. Apply slow, continuous pressure for two or three minutes. With a bur the pulp should now be exposed, and, if still found sensitive, the process is to be repeated. As an anesthetic solution for this purpose, a 5 per cent solution of novocain in sterile water is recommended. Or a wisp of cotton saturated with the same solution or a moistened novocain pluglet is placed in the hole, covered with dry cotton and a piece of very plastic unvulcanized rubber and pressure is made with an instrument that will just about fit into the opening.

Within recent years a number of complicated syringes, variously known as high-pressure syringes or obtunders, have been advocated,

based upon the principle of forcing anesthetic solutions through sound tooth substance with intense pressure. This conception of pressure anesthesia is erroneous. Close contact of the anesthetic fluid with the dentinal fibers, plus the necessary time for conveying the absorbed anesthetic *via* the Tomes' fibers to the nerve endings, explains the phenomenon very plausibly. A strong metal syringe, provided with a specially prepared needle to make as near as possible a water-tight joint, is all that is required. Those who prefer a special high-pressure syringe for such purposes may purchase any one of the many devices that will suit their fancy. The Weaver obtunder or the Jewett-Willcox syringe is much lauded for such purposes. Any one of the various methods for anesthetizing a tooth as outlined under "Local Anesthesia as Applied to Operative Dentistry" may also be used for anesthetizing the pulp.

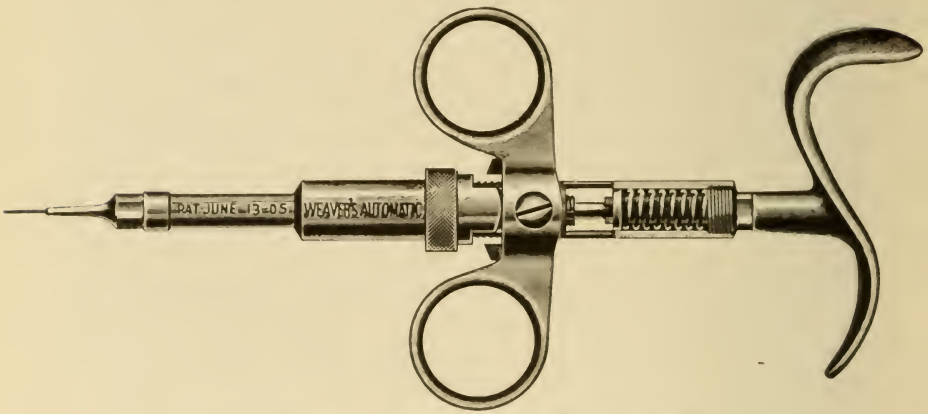


FIG. 73.—Weaver high-pressure obtunding syringe.

In teeth not fully calcified and in so-called "soft" teeth, pressure anesthesia produces most satisfactory results, while the process is applied with difficulty in teeth of elderly persons, teeth of inveterate tobacco chewers, worn, abraded and eroded teeth with extensive secondary calcific deposits, teeth whose pulp canals are obstructed by pulp nodules, teeth with metallic oxids in their tubules, teeth with leaky old fillings, badly calcified teeth, mainly all from one and the same cause, namely, clogged tubules. In most cases no amount of persistent pressure will prove successful.

According to Hertwig the protoplasm of the cell primarily transfers irritation and, secondarily, transmits absorbed materials, and therefore the anesthetic solution has to pass through the entire length of the dentinal fibrils before the nerve endings of the pulp proper are reached. Consequently a certain period of time is required before the physiologic effect of the anesthetic is manifested, and this period of latency is dependent on the thickness of

the intermediate layer of dentin. The successful anesthetization of the pulp depends largely on this most important factor of allowing sufficient time for the proper migration of the drug.

Other soluble local anesthetics, such as nervocidin, erythrophlein hydrochlorid, quinin and urea hydrochlorid, etc., have been advised at various times as reliable pulp anesthetics. Owing to numerous drawbacks, these drugs have never obtained popularity. Refrigerant local anesthetics have also been advised for the extirpation of the pulp. The application of this group of anesthetics for such purposes is usually accompanied by many disadvantages which materially limit their usefulness.

Destruction of the Pulp by the Actual Cautery or a Potential Caustic—Devitalization of the pulp by the actual cautery or a potential caustic has been in the past more freely used than any other method. Before entering into a discussion of the various procedures employed in their application, it is probably not amiss to rehearse their pharmacologic action.

Caustics, sometimes called escharotics, are substances which destroy living tissue by virtue of their coarse chemical or physical action, affecting organized as well as unorganized albumin. The older medical lexicographers restricted the term escharotic to substances which produce a dry, more or less insoluble, protective slough. They further differentiated between the actual cautery, *i. e.*, the red-hot iron, and the potential cautery, *i. e.*, an agent, like silver nitrate, which forms an eschar without the agency of actual fire.

True caustic action manifests itself essentially in two definite ways: It produces coarse chemical or physical changes in the tissue which are macroscopically recognizable or it causes more or less direct death of those affected tissues. Pure chemical drug action on living cell structures which endangers, or even kills, the cell without visible changes is referred to as protoplasm poisoning, while a drug which produces severe visible tissue changes, but without cell destruction, is spoken of as an irritant. A concrete example of differentiation between a caustic and a protoplasm poison is readily furnished by a comparison of the action of the following two drugs: If a dram of pure sulphuric acid is swallowed it will, by cauterization, destroy the tissues with which it comes in contact and thereby finally kills the individual. If the same amount of acid is dissolved in a quart of water and drunk, it will practically do not visible harm as it is now so very diluted as to be inactive. If, on the other hand, a protoplasm poison, as for instance, 3 grains of arsenic, are taken as a dose, whether it is in the form of a powder or dissolved in a quart of water, it will always kill a man, no matter how well diluted.

In the early days of the practice of conservative dentistry, as Fauchard, Bourdet, Hunter, Koecker and others inform us, the

actual cautery was freely employed for destroying the "nerve" of a tooth. The method utilized for this purpose was practically the same as that employed by Koecker for "burning the nerve" (p. 104). At present the electric cautery is very rarely employed, while the potential caustics, such as nitric acid, silver nitrate, "spirit of salt" (hydrochloric acid), which were also recommended in bygone days, have only a limited usefulness. In 1833 Wood advocated the use of crude arsenic (flystone, ratsbane or native cobalt bloom)[1] for the destruction of the dental pulp. Three years later, in 1836, Shearjashub Spooner,[2] of New York, published an excellent little book entitled *Guide to Sound Teeth, or a Popular Treatise on the Teeth*, in which he recommended to the dental profession for the first time the use of arsenic trioxid for the above purpose. It is stated that Chaplin Harris, of Baltimore, used arsenic in 1835 without having knowledge of Spooner's discovery.

The original description of the introduction of arsenic trioxid for the destruction of the dental pulp by Spooner is, from a historical point of view, most interesting and is here appended in outline.[3]

[1] Crude cobalt, better known as flystone (Scherben Kobalt) is an impure cobalt arsenate containing variable quantities of arsenic. The pure metal cobalt is, pharmacologically, an inert substance; in its crude form its action depends upon the presence of arsenic.

[2] Spooner, Shearjashub: Guide to Sound Teeth, 1836.

[3] Treatment of the Teeth when Caries Has Progressed to the Nerve.

When decay has extended to the nerve and causes the tooth to ache extraction is the usual remedy resorted to by all who have sufficient resolution to induce them to undergo the operation. The teeth are so liable to caries in this country, and often decay at so early an age and so rapidly, that it is a matter of very great importance to all, especially to those who have been so unfortunate as to lose many teeth, whether some means cannot be devised for their preservation. This desideratum has been happily discovered, and its success has been thoroughly tested, in a practise of many years. The nerves of the teeth may be certainly and effectually destroyed, with little or no pain to the patient and without the least danger, by means of a little arsenious acid (arsenic, ratsbane) applied to the nerve. We claim for our brother, Dr. J. R. Spooner, of Montreal, the credit of this valuable discovery and for ourselves no small share of credit for thus frankly laying it before the dental profession and the public. We have proved that the vitality of the fangs of the teeth, which is necessary to prevent them from acting as foreign bodies in their sockets, does not depend upon the internal membrane or nerve. This fact is of greater importance than one would suppose at a first view, for thousands of aching teeth which are daily being extracted may be effectually preserved by taking advantage of it. This is a matter of much moment to those who have been unfortunate as to lose many teeth. So complete and satisfactory is the operation of arsenic in destroying the living fiber, that instead of extracting teeth whenever the nerve is badly exposed, we destroy it, plug the tooth and thus preserve it. Teeth thus treated will often last a great many years and prove highly serviceable. Arsenic will not only positively destroy the nerves of the teeth, but it possesses the great advantage that it does this without causing pain. If it be applied to an aching tooth it slightly augments the pain, but when applied to a nerve, not inflamed, it does not cause any pain worth minding. Of late we have applied to aching teeth a mixture composed of arsenic, 3 parts, and acetate of morphine, 1 part, the morphine being the most powerful odontalgic remedy that we know of. Thus the most violent toothache may be effectually cured without additional pain, a circumstance well worth considering. We cannot too strongly recommend this treatment to the public, under the circumstances we are treating of.

The specific action of arsenic on the tooth pulp may be epitomized as follows: If applied to an exposed normal pulp it is readily absorbed. Pronounced hyperemia and consequently increased pain are the early manifestations of the arsenic action. The vascular system is most readily affected by it and very pronounced dilation of the vessels, especially the capillaries, with more or less stasis of the circulation is the outstanding feature of its initial effect. "The walls of the capillaries are exceedingly delicate, being formed by a single layer of endothelium, which is a continuation of the endothelial lining of the arteries on the one side and the veins on the other" (Hopewell-Smith). The endothelial coat of the capillaries is quickly corroded, causing multiple hemorrhages. Destruction of the blood plates, *i. e.*, plasmolysis and plasmorhexis, immediately follows, resulting in granular detritus. Thrombosis and stasis are the direct sequences. The connective-tissue fibers and the odontoblasts are but little altered. The primary point of attack on the nerve centers is located in their endings, causing a destruction of the myelin and a more or less pronounced neuritis results; the latter is usually followed by complete cessation of all pain. The pronounced disturbances of nutrition finally result in anemic collapse and shrinkage of the entire pulp mass.

On pathologically altered pulps aresenic acts very much in the same manner; its action is somewhat increased in the early forms of pulpitis, but usually slower in those cases in which severe inflammation and suppuration is predominating. An existing neuritis is always markedly increased. Depending on the vascularity and the size of the pulp, and the quantity used, from a few hours to two to five days are usually required for its progressive destruction. Strangulation of the pulp about its apical end, resulting from the intense hyperemia, brought about by the action of arsenic, is not

No selfish views evidently actuate us in making these declarations. They are not the puffs of pretenders. Arsenic is the only substance with which we are acquainted, that will effectually destroy the nerve of a tooth. Nitric acid and nitrate silver have been much employed for this purpose. They were favorite toothache remedies with Hunter and Abernethy, but they do not generally do this effectually; they only destroy the surface of the nerve; indeed but the small portion of it is exposed; besides they destroy the tooth. A hot wire is the remains of barbarism, cruel as death and does not become the present enlightened day. A drill, as well as the hot iron, is very painful, and is enough to strike horror to the soul of a patient. The arsenic affects the object like a charm, and under proper management never fails of complete success. Many persons, of course, would be apprehensive of danger from its use; $\frac{1}{20}$ grain is quite enough to destroy the nerve of any tooth. But let no ignorant person dabble with this remedy. As we recommend it so strongly, it is a duty incumbent on us to caution the public. There are many who would have no hesitation in filling a large hollow tooth with arsenic, on the strength of this recommendation. There are some who do not know even what it is! We know of one instance, and but one, thank Heaven, in which the application of the remedy was attended with fatal consequences, and we merely mention it as a warning to presuming quacks. We have used this remedy in hundreds of instances, without ever experiencing any ill effect; nor can any danger ever attend its proper application. Whenever the nerve of a tooth can be preserved it should not by any means be destroyed.

the direct cause of its death; in teeth with undeveloped or in those with partially absorbed roots, strangulation is very doubtful.

Since the introduction of arsenic trioxid for the purpose of destroying the dental pulp many substitutes have been advocated, but none have so far superseded it or taken its place. Aside from the application of local anesthetics by special methods, arsenic trioxid is still the most universal agent employed for the above purpose. Usually it is applied in the form of a paste, sometimes as arsenical fiber or disks and as a dry powder. Innumerable formulas for compounds of arsenic with other drugs are suggested for such purposes. The principal object has always been to com-

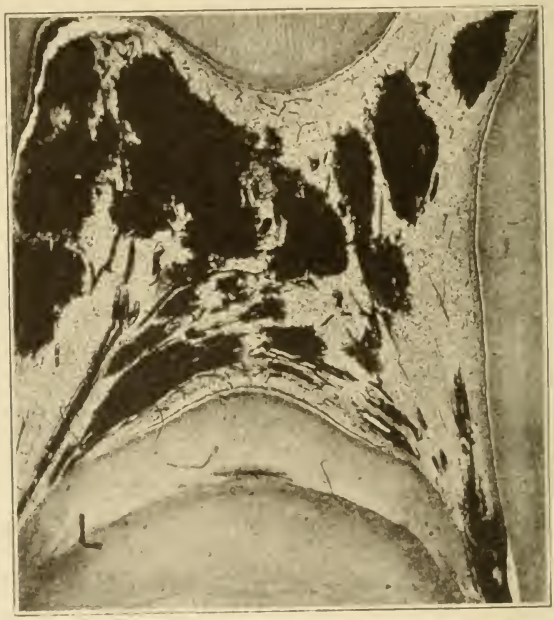


Fig. 74.—A pulp in a molar devitalized with cobalt. (Moginier.)

bine arsenic trioxid with an anesthetic. Many of the published formulas represent empirical compounds, which are put together in utter disregard of the pharmacologic action of the individual drugs. If the pulp is in a normal condition, very little or no pain is manifested by the arsenical application; if the nerve cells are inflamed or are undergoing necrobiotic changes the increased irritation brought about by the powerful oxidation and reduction as a result of the pharmacologic action of arsenic increases the already existing neuritis, and more or less severe pain results. Arsenic is very diffusible; it quickly destroys the nerve endings, and consequently there is little chance for the anesthetic which may be

added to it to exercise its specific function. For this very reason the addition of a local anesthetic is of no benefit.

For many years the original formula of Spooner, consisting of arsenic trioxid, morphin acetate and creosote, has been and is still used with apparent good success. Morphin applied locally has no anesthetic or narcotic effect on sensory nerve endings, and consequently it acts merely as a diluent of the arsenic trioxid. Cocain or its substitutes added with the expectation of mitigating the pain or the irritating effect of the arsenic trioxid is, to say the least, questionable. Scientific proof of this supposition has certainly never been brought forward. Nevertheless, there is less objection to their use than to most of the other narcotics. Additions of aconite, eserin, opium or its alkaloids, iodoform, etc., are useless, as they simply interfere with the ready absorption of arsenic. A

Fig. 75.—Devitalized pulp. Magnified section of Fig. 74. (Moginier.)

more rational procedure consists in applying to an aching pulp a concentrated solution of a local anesthetic prior to the application of the arsenical paste. It is claimed by Lipschitz that the application of liquid phenol to the exposed pulp for at least ten minutes prior to the placing of the arsenical dressing will prevent pain arising from the action of the devitalizing compound.

The addition of an antiseptic to the arsenical paste is illogical. Arsenic is a powerful antiseptic in itself, although it is well known that the cell walls of the lower organisms (bacteria) possesses a greater resistance to its action than those of the higher organized cells. Tanning agents are frequently added to the paste for the purpose of changing the pulp tissue to a leathery material, so as to facilitate its ready removal. Tannic acid or the various forms of formaldehyd are used for this purpose. It is better practice to

apply such agents after the arsenic dressings has been removed; the less we interfere with the absorption of the arsenic, the better and quicker will be the results.

As a vehicle for the paste, only such media as are more or less solvents of arsenic trioxid, or which allow its ready absorption by the pulp, are justified. Glycerin, lanolin, vaselin, phenol, creosote, or the essential oils, and similar liquids, have been used for many years as vehicles for the paste; their influence on the action of arsenic is apparently of very little consequence; they certainly do not exercise their typical pharmacologic action in this connection. Strong coagulants should not be used as they hinder the ready absorption of the poison by forming a scab. To give a distinct color to the paste, very small quantities of lamp black or carmin may be added. Some practitioners prefer to apply arsenic in the form of a paste mixed with cotton fibers, or in the form of paper disks saturated with a soft paste. Arsenical fiber is prepared by mixing cross-cut cotton with the paste, and the disks are made by saturating very small square of hard white blotting paper with the thin paste, which are then dried and preserved.

Prior to the application of arsenic, the cavity must be excavated, as arsenic will not act through disorganized dentin, and, if possible, the pulp should be exposed and thoroughly depleted, either by puncturing the organ or by applying vasoconstrictor drugs. Lavage has been recommended for this purpose, *i. e.*, washing the pulp with lukewarm water, changed slowly to cold water. Quicker results are, however, obtained by applying epinephrin chlorid solution under pressure. The cavity must be free from blood to prevent the formation of inactive arsenic hemoglobin. If the pulp is inflamed and painful it is absolutely necessary to apply suitable sedative remedies to relieve the conditions before the paste is applied; an inflamed pulp materially hinders the ready absorption of arsenic, and continuous severe pain is certain to follow. A solution of novocain or chloretone in oil of clove is serviceable for this purpose. These remedies, if sealed into the cavity, usually alleviates the condition in from twenty-four to forty-eight hours. If pus is present it must be drained off. Pulp nodules occasionally obstruct the ready diffusion of the chemical. Removal of these calcareous deposits by means of a drill, after pressure anesthesia has been applied, is indicated. Cocain should never be applied cataphorically under these conditions, as the electric current may drive the previously applied arsenic through the apical foramen into the soft tissues. Occasionally one meets a patient who presents an unexplained idiosyncrasy to the action of this chemical.

The cavity for the reception of the arsenical application should be of ready access, and so prepared as to easily retain the temporary filling. The arsenical compound is preferably placed in direct

contact with the freely exposed pulp by means of a blunt instrument, or on a depressed metallic disk or a piece of cardboard, or on cotton or spunk. Close contact insures quick action. Arsenic will act by osmosis, although slower, through any thickness of dentin. This very fact is the reason why its use as a remedy for hypersensitive dentin has been abandoned; death of the pulp is invariably the sequence of such procedures. Some operators prefer to cover the arsenical dressing with an intermediate film of plain or oiled paper, or a pledget of cotton.

The final sealing of the cavity consists of a temporary filling of cement or of a gutta-percha preparation. Extreme care should be exercised in this simple, yet most important, operation. Cotton fibers mixed with sandarac or mastic varnish, to be used as a retaining medium, should be avoided; they readily become foul in the fluids of the mouth, or they may leak, and, besides, they swell, causing pain from pressure on the pulp. Kirk has advocated the use of surgeon's rubber plaster where but a portion of the tooth is left, carrying it around the tooth; it will adhere satisfactorily for several days or long enough to accomplish the object. The gutta-percha preparations are the best media for a temporary dressing seal; most experienced operators agree that a cavity correctly sealed with this material offers less possibilities for the seeping through of the drug than the various cements, etc. In applying the temporary stopping it is very essential to avoid pressure on the dressing. In proximal cavities, where overhanging tooth substance prevents ready access, and therefore presents danger of misplacing the arsenical dressing, gutta-percha packed between the two teeth is of service.

The quantity of arsenic necessary for the destruction of a pulp is very small. A careful estimation based on diverse weighings of quantities of arsenical paste, as employed by several practitioners in their routine work, has shown that the average application weighs about $\frac{1}{30}$ grain (0.002 gm.). It is not only useless, but decidedly dangerous, to employ more. Other writers have estimated the amount as varying from $\frac{1}{100}$ to $\frac{1}{20}$ grain.

In deciduous teeth, and in those of young persons where the roots have not fully formed, the arsenical paste should be left in the cavity only a few hours. Many practitioners are opposed to its use in the teeth of children. More than two teeth should not be subjected to the treatment at one sitting, to prevent a possible chance of an accidental swallowing of a large amount of the poison.

The time required for the destruction of the pulp with arsenic depends on many circumstances. In the young, on account of the vascularity of the organ, from four to eight hours are usually sufficient. In people of mature age it is best to leave the application *in situ* from three to four days. This allows ample time for

the breaking down of the entire pulp and its ramifications. Many pulps do not, however, require more than one or two days to succumb to the effects of the poison. After the arsenic has been removed it is well to seal an astringent into the cavity for twenty-four hours, it will greatly facilitate the ready removal of the pulp *in toto*. Occasionally it will be found that in the removal of the pulp the apical half is still very sensitive to the touch. If it becomes necessary to again apply arsenic in the root canal a very small quantity of the paste carried on the end of a barbed broach which is quickly thrust into the pulp stump, should be employed.

The following important facts should be remembered when an arsenical compound is used for the purpose of destroying the pulp:

1. Only the smallest possible quantity which will kill the pulp should be used.
2. Arsenic should never be applied on a severely aching pulp.
3. On teeth with partially absorbed or with undeveloped roots the arsenical paste should remain only from four to six hours.
4. If possible, the paste should be applied on a freely exposed and depleted pulp.
5. A retaining seal must be applied without pressure and with the utmost care.

Local toxic effects in the mouth are most frequently met with as the result of faulty application of the chemical for dental purposes. Leakage of the dressing seal is responsible in most cases, and contact of the mucous membrane with instruments accidentally carrying small particles of the paste, or the unnoticed squeezing out of arsenic resulting from pressure applied on placing the retaining stopping, are possible factors. The fact that arsenic trioxid is odorless and tasteless increases this danger, which usually is recognized only after the mischief is done. Numerous cases of severe forms of toxic periostitis, followed by necrosis of the alveolar process, and loss of one or more teeth, are on record.

Lateral passage of arsenic or any other drug through the dentinal tubules and the cementum into the pericementum does not occur. The homogeneous, structureless layer of cementum, by virtue of its position, acts as an efficient barrier to the outward passage of drugs placed in the root canal.

Arsenical intoxication of the gum tissue presents in its early stages all the phenomena of true inflammation. Later the surfaces become denuded and assume a raw ham color; the veins are distended, the border of the infected area is raised and shows a loss of substance in the depressed center—the typical picture of an ulcer. Usually there is a pronounced metallic taste present in the mouth. Arsenic penetrates very deeply, destroying the soft and hard tissues, which finally results in true necrosis. In the early

stages the affection is not painful, but as soon as the deeper structures are reached severe pain is manifested.

The treatment depends on the severity of the poisoning. Simple intoxication requires the immediate removal of the cause and mild antiseptic mouth washes. If necrosis has set in the affected parts must be thoroughly curetted with a large spoon excavator; if the bone has been sequestered it must be removed. Local anesthesia is usually serviceable for such work. The denuded surface is covered with a suitable surgical dusting powder. Rigid antisepsis is of prime importance. A warm physiologic saline solution used at frequent intervals is indicated as a mouth wash. The local application of dialyzed iron as an arsenical antidote is indicated only if arsenic is present in substance on the tissues; after it is absorbed this solution is useless.

DEVITALIZING PASTE.

℞—Arsen. trioxid . . . . . gr. xxx (2 gm.)
Ol. caryophyl . . . . . q.s. to make a paste
Add a small amount of lampblack to color the paste.

DEVITALIZING FIBER.

℞—Arsen. trioxid . . . . . gr. v (0.3 gm.)
Acid. tannic . . . . . gr. ij (0.12 gm.)
Phenol. liquefact . . . . . q.s. to make a thin paste
Sig.—Fine cross-cut absorbent cotton fiber is mixed with this paste and dried.

DEVITALIZING DISKS.

℞—Arsen. trioxid . . . . . gr. xvj (1 gm.)
Ol. caryophyl . . . . . q.s. to make a thin paste
Sig.—Cut small squares (1 to 1.5 mm.) of hard white blotting paper, saturated with the paste, dry in the open air and then put into a glass-stoppered bottle.

Removal of the Pulp.—The removal of the anesthetized or devitalized pulp should always be done under strict aseptic conditions. The preparation of the field of operation, the sterilization of the instruments and the general technic of the procedure is practically the same as that employed in the removal of gangrenous pulp débris as discussed under "Necrosis and Gangrene."

To facilitate the disintegration of the devitalized pulp prior to its extirpation, pepsin or its substitute, papain, have been advocated as a means of digesting the dead tissue. Oakley Coles, Arkœvy and Harlan have recommended these agents for such purposes; they are rarely employed at present. The following combination was advocated by the late Harlan:

℞—Pepsin . . . . . gr. v (0.3 gm.)
Acid. hydrochlor. dil. . . . . gtt. j
Glycerin . . . . . q.s. to make a stiff paste

Pack the paste into close contact with the dead pulp, seal the cavity and leave undisturbed for a week. The dead pulp will usually be found digested (liquefied) within that time.

CHAPTER XVI.

MUMMIFICATION OF THE DENTAL PULP.

Clinical evidence confirms the statement that dead pulp tissue when left in a root canal without being rendered permanently sterile will sooner or later cause disturbances from decomposition with all its sequels, as a result of secondary bacterial invasion. Infective microörganisms may have been present in the diseased pulp; they may have been introduced at the time of treatment, or they may have reached the pulp tissue through the apical foramen *via* the blood current. Faisztl has experimentally demonstrated that hematogenous infection of the dental pulp is possible.

Soon after the discovery of creosote in 1830, as Flagg informs us, the dentists tried this agent for the purpose of preserving dead pulps left *in situ*, while Flagg himself employed a paste composed of phenol, oil of clove, sulphate of lime and acetate of morphin. In 1874 Adolph Witzel made an effort to solve the problem of pulp mummification by advocating the amputation method which was first practised by the late W. W. Allport. Witzel supposed that arsenous acid when placed upon an inflamed pulp would only destroy the diseased portion of this organ, while the remaining part, when separated by a slough, would preserve its vitality. The pulp stumps left in the canals were capped with an iodoform or a phenol cement of various compositions. In 1886 he changed his views and he referred to the pulp stumps as "being shrunken to antiseptic threads," etc. Telchow, in 1882, announced that he had found in Wickerscheimer's preserving fluid a medium with which he could successfully mummify dental pulps. This fluid is a complex mixture of alum, arsenic, various inorganic salts, methyl alcohol and water, and is used as a preservative for anatomic specimens. In 1888 Baume recommended borax for the purpose of embalming pulp stumps. The borax method, due to the very weak antiseptic power of this chemical, was a complete failure. In 1892 Herbst announced that he had found a method of pulp amputation which was simplicity itself. He placed upon the exposed pulp a mixture of 92 parts of native cobalt (crude cobalt arsenate) and 8 parts of cocain hydrochlorid. After a few days, the coronal portion of the pulp was removed under aseptic precautions, the cavity sterilized and tightly closed with soft tin, burnished into place by his rotation method. This is in reality the old way of treating pulps

with arsenic as practised by our fathers. Even as late as 1888, Cunningham placed a pledget of cotton saturated with a mixture or arsenous acid in alcohol and oil of clove in the root canal of the molars for the purpose of preserving the pulp stumps. For a few years, teeth treated by this method would be quiescent, but after about five years 50 per cent or more had abscessed. In 1892 the late W. D. Miller suggested a mixture of 3 parts of corrosive sublimate and 1 part of thymol, compressed into small tablets which he used in about the same manner as Baume recommended for his borax embalming process. The success obtained with these tablets has been fairly satisfactory, the bluish-black discoloration of the teeth by the sublimate and the very frequent acute pericementitis following the treatment are serious objections. Sœder-

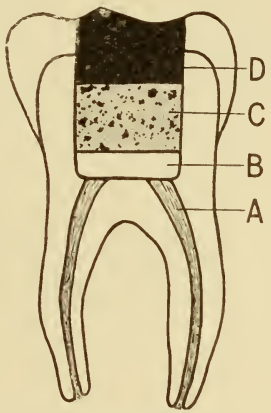


FIG. 76.—Schematic drawing of pulp-mummification. *A*, pulp stumps; *B*, mummifying paste; *C*, cement filling; *D*, amalgam filling.

berg published an interesting account of his pulp mummifying process in 1895. He prepared a mixture of equal parts of thymol, alum and glycerin with enough zinc oxid to make a stiff paste. This paste has been employed with more or less success and from 1896 to about 1902 the current literature is filled with reports and discussions on this interesting question. In 1898–1899 Bœnnecken recommended a method of pulp mummification, using a paste consisting of cocain, thymol, formaldehyd, zinc oxid and glycerin. The success obtained with this treatment was apparently satisfactory, and his method has gained many friends. Brooks, in 1898, offered chromic acid in conjunction with sulphuric acid for such purposes. In 1898 Julius Witzel published his experiments with formaldehyd and sulphuric acid, and a year later Lepkowski recorded his studies on the action of formaldehyd on pulp tissue.

Gysi, in 1899, experimented with a large number of drugs to be used as pulp stump preservatives and he finally arrived at the conclusion that the successful mummification depended upon the combination of an easy and a difficult penetrating antiseptic. Preiswerck, in 1901, advocated a mixture of borax and eugenol with the addition of small quantities of tannic acid. Since then, a host of other publications have appeared, and especially since Gysi advocated a mixture of formaldehyd and cresol for the treatment of infected root canals, this very mixture has also been recommended as a pulp mummifier. A most interesting monograph on this subject has been written by Bœnnecken, in which he treats the subject of pulp mummification from the clinical as well as from the experimental point of view.

Pulp mummification has been advocated for such cases in which it is impossible to remove all of the pulp tissue. It should be remembered that this statement refers only to organized pulp tissue, viz., the pulp may have been in a normal state of health or in any stage of pulpitis except complete suppuration, prior to its devitalization, but it must never be employed upon a gangrenous pulp. It is an important dictum that a pulp suitable for this method of treatment must be recently devitalized by a caustic. It goes without saying that pulps which have been temporarily anesthetized by pressure or contact anesthesia have to be devitalized by a caustic before becoming eligible to the process.

It is an imperative law of modern conservative dentistry that all available efforts should be made by the conscientious practitioner; to remove, if possible, all of the dead pulp tissues. Even if we are able to mummify a devitalized pulp in its entirety, this method should not be practised indiscriminately as it is antagonistic to one of the fundamental concepts in surgery which teaches us to remove all necrosed tissue, and its general practice may create a tendency to carelessness and imperfect work on the part of the operator. As a consequence, this method of treatment has been discarded at present by the practitioners in the United States. The chief objection raised by the advocates of the mummifying principle against the generally accepted method of complete extirpation hinges primarily on the supposed inability on the part of the operator to remove all of the pulp tissue from certain root canals due to their peculiar anatomic structure. This objection is largely nullified by the intelligent use of a properly selected instrumentarium in conjunction with well-defined roentgenograms of the respective tooth. The objection to the expenditure of time as required for this tedious operation should not receive any consideration by the conscientious practitioner. However, in such very rare cases where it is utterly impossible to remove all of the pulp tissue, *i. e.*, principally in multi-rooted teeth, and where it is

essential to save the tooth, the application of the mummifying principle may be justified. The pulps of the deciduous teeth are especially amenable to this mode of treatment.

The process of mummification of pulp tissue is based upon certain fundamental principles which have to be fully recognized in order to insure success. Wrong diagnosis of prevailing conditions of the pulp, faulty technic in the application of the various medicinal agents and the use of inadequate drugs are largely responsible for the failure obtained with this procedure. Without entering into a detailed enumeration of the various compounds which have been used experimentally, let it suffice to say that thymol answers this purpose well. It is a phenol-like body of greater antiseptic strength than pure phenol and is only very slowly soluble in water. As this compound does not possess enough "body," some inert material should be added thereto, such as zinc oxid or a bismuth salt, etc., and enough cresol or some other suitable fluid antiseptic to form a substantial paste. A suitable mummifying paste may be prepared according to this formula:

Thymol	10 parts
Cresol	10 "
Zinc oxid—enough to make a stiff paste.	

The thymol is dissolved in the cresol by trituration and enough zinc oxid is added to make a stiff paste.

Gysi's *triopaste* has the following composition:

Cresol	10 parts
Creolin	20 "
Glycerin	4 "
Trioxymethylen	20 "
Zinc oxid	66 "

As we have stated above, only such cases are amenable for this method of treatment in which the pulp is not gangrenous. The pulp is devitalized with arsenous trioxid in the ordinary way. The latter should be left in position for about two to three days, depending upon the vascularity of the pulp. Under the strictest aseptic precautions the coronal portion of the pulp is drilled out with a round bur and an effort is made to remove from the canals as much of the pulp tissue as possible. The cavity is repeatedly flushed with sterile water and dried with bibulous paper.

A concentrated solution of (50 per cent) zinc chlorid in distilled water is now allowed to flow into the root canals so as to thoroughly impregnate the pulp remnants. After a few minutes the excess fluid is removed with sterile paper cones and a small quantity of mummifying paste is worked into each canal, pressed into position with a tightly rolled pellet of cotton, and the remainder of the root canal and the pulp chamber are filled with cement.

If the aseptic technic of pulp mummification is applied strictly according to the above discussed *modus operandi* and if careful attention is paid to the correct diagnosis of the existing condition of the pulp, success will in the majority of cases be assured. Experimental work as carried out by Cuendet[1] with Gysi's "triopaste" revealed the fact that within a period of from four to ten months in 92 per cent of the cases the root canals remained sterile. No disturbances of the periapical tissues were observed. However, we should be mindful of the fact that observations extending over a period of ten months do not furnish sufficient proof to render final judgment about a therapeutic procedure of the above discussed nature; at least five years' time and many individual cases are required to substantiate clinical results.

[1] Inaugural Dissertation, Zurich, 1920.

CHAPTER XVII.

NECROSIS AND GANGRENE OF THE DENTAL PULP.

Synonyms.—Death and putrefaction of the dental pulp, *necrosis et gangræna pulpæ.*

Definition.—Death of the dental pulp from any cause and simultaneous or subsequent infection resulting in putrefaction.

Etiology.—Death of the pulp may be caused by any one of the numerous disturbances as outlined under Etiology, or it may be artificially produced by a toxic agent, usually arsenic trioxid. Gangrene of the pulp is the result of secondary putrefactive changes occurring in the necrotic tissue. Necrosis and gangrene are primarily caused by a bacterial invasion, usually a streptomycosis of a mixed type, arising as a sequence of existing dental caries.

Varieties.—Clinically, necrosis and gangrene of the dental pulp may manifest themselves as any one of the numerous varieties as discussed below.

Clinical Pathology.—The clinical manifestations of necrosis and gangrene depend primarily upon the respective state of the dead pulp. True necrosis without infection in an otherwise sound tooth usually presents no visible signs of disarrangement, except a possible discoloration of its crown and it causes no inconvenience to the patient. If the pulp chamber is exposed putrefaction of the dead pulp will reveal itself to the patient by foul odors and a bad taste. As soon as the products of the putrefactive process penetrate into the periapical tissues, marked disturbances arise which manifest themselves in the majority of cases as an acute alveolar abscess which at some future period may assume a subacute or a chronic type. If the periapical infection is of a very mild type the vital reaction of the disturbed pericementum institutes a process of defense resulting in a chronic proliferating pericementitis, usually referred to as a granuloma. Should this granuloma contain epithelial cells, the mild irritation may cause this latter tissue to proliferate in all directions and finally form a complete capsule. Degeneration of the newly formed epithelial goblet cells occurs and their contents, *i. e.*, mucus, fat and other products of decomposition collect in the interior of the granulomatous sack and by pressure from the accumulated fluid the surrounding bone is absorbed and a radicular cyst is the usual sequence.

A clear conception of the chemistry and the bacterio-pathology

of the various manifestations as they occur in dead pulp tissue is of the utmost significance as related to the future treatment of the disturbance, hence a detailed discussion of this momentous phase of the pathology of infected root canals is of vital importance to the clinical practitioner.

The dental pulp is composed of connective tissue, nerves and bloodvessels. All animal tissues are essentially built up of cells and the constituents of these cells consist of proteidogenous matter, lipoids, salts and water. Only a very few elements enter into their make-up, *i. e.*, nitrogen, oxygen, carbon, hydrogen, sulphur and very little phosphorus and iron. The tissues containing nitrogen are referred to as nitrogenous substances, or proteins, while non-nitrogenous substances are spoken of as carbohydrates and

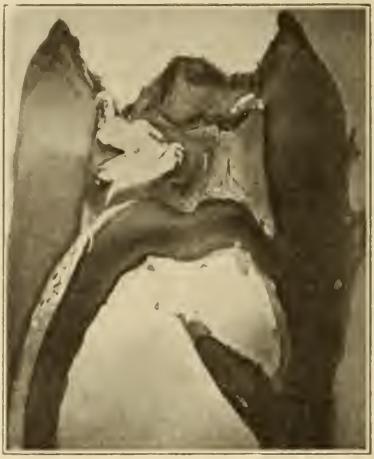


FIG. 77.—Gangrene of the dental pulp.

fats. The normal pulp tissue is composed principally of proteidogenous material, and so far no carbohydrates or free fats have been isolated from it. The proteins are the most complex bodies known to chemistry; they are usually colloidal in their nature, and are composed of molecules that differ widely in their weight and size. The average protein molecule approximately furnishes the following constituents: Carbon, 51 to 55 per cent; oxygen, 20 to 24 per cent; nitrogen, 15 to 17 per cent; hydrogen, 6.8 to 7.3 per cent; sulphur, 0.3 to 0.5 per cent and very small quantities of phosphorus and iron. The proteins may be decomposed by acids, alkalies and ferments. These latter substances are found extracellularly in the digestive juices and intracellularly where they become manifest in autolysis; they are produced by bacteria. In the decomposition of the pulp we are principally concerned with

the intracellular (autolytic) and bacterial enzymes. The ferments which are secreted by the leukocytes during the various inflammatory processes within the diseased pulp are the digestive agents, as Kantorovicz has shown. Death of the pulp, *i. e.*, necrosis, is the precursor of pulp decomposition, *i. e.*, gangrene.

Whenever healthy tissue becomes irritated by physical or chemical (including bacteria) agents to such an extent as to cause intense disordered cell nutrition, death of the cells results. This process is known as necrobiosis. A pulp may accidently die of its own accord through any of the above causes, or it may be intentionally killed by a caustic, usually arsenic trioxid.

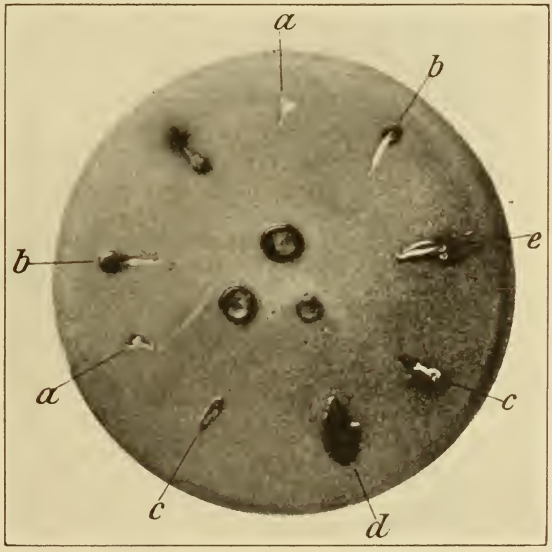


FIG. 78.—Action of ferments produced by the leukocytes in diseased dental pulps (Kantorovicz): *a*, healthy pulps; *b*, pulps with partial acute pulpitis; *c*, pulps with total acute pulpitis; *d*, pulp with supurative pulpitis; *e*, pulp with an abscess containing pus. The center contains granulomas obtained from roots.

In general pathology four varieties of necrosis are usually recognized:

1. *Coagulation Necrosis.*—This form of necrosis results from the coagulation of fluids that have entered into or are present in the pulp, and that contain coagulable substances, *i. e.*, the soluble colloidal material is transformed into insoluble modifications. The change of fibrinogen into fibrin is an important factor in this procedure. The pulp assumes a dry, firm appearance, and is usually of a yellowish color. When blood enters into the root canal after the removal of a coagulated pulp, it usually becomes quickly clotted. Coagulation necrosis may be caused by heat, phenol,

corrosive sublimate and other chemicals, and it is rather seldom met with in the ordinary cases of death of the dental pulp.

2. *Liquefaction or Colliquation Necrosis.*—This type of necrosis occurs principally in the central nervous system; its etiology is not quite clear, and probably edematous infiltration and enzyme action have much to do with it. It may be caused by the action of chemical substances resulting in aseptic suppuration. True suppuration should not be confounded with it. It is very rarely observed in the dead pulp.

Fig. 79.—Gases formed by a putrid pulp upon culture medium.

3. *Caseation Necrosis.*—This term is applied to a type of coagulation necrosis which resembles an emulsion of fat and water, and has the appearance of soft cheese. The coagulum is made up of protein derivatives, considerable quantities of fat and water, etc. Fatty degeneration of the pulp as a whole is rarely observed. The action of proteolytic enzymes is probably largely responsible for these changes. Caseation is most frequently found in pulp decomposition.

4. *Fat Necrosis.*—This specific type of necrosis is restricted to fat tissue and is characterized by a splitting of the fat into fatty acids and glycerol. As the normal pulp does not contain free fat, fat necrosis is probably never observed in its pure form in this organ.

Gangrene.—Gangrene is the result of secondary putrefactive changes, and it results from the dual action of proteolytic enzymes and putrefactive organisms. Two forms of gangrene are usually recognized in general pathology, *i. e.*, moist and dry gangrene. Moist gangrene depends on the presence of water, while the absence of water denotes dry gangrene or mummification. In dry gangrene nearly all further changes cease, while in the moist form the autolytic changes continue. A totally gangrenous pulp presents a mass of débris in which lime concretions, fat droplets, crystals of fatty acids, of hematoidin and of triple phosphates, numerous bacteria, and various pigments are the only discernible elements. The fat droplets are partially produced by fatty degeneration of the myelin sheaths of the nerve fibers and partially by disintegration of the cell protoplasm and dead bacteria. The latter organisms apparently contain fat as a metabolic constituent in the form of lipoids. In the great majority of cases of pulp disintegration progressive moist gangrene is predominating. In clinical practice complete moist gangrene of the pulp is not always

found, and the latter organ may be partially or totally gangrenous. In partial gangrene one part of the pulp may be totally putrescent, while the other part may be still in a state of severe inflammation. A fairly distinct line of demarcation may be observed between the dead and the inflamed part of the pulp. Through necrobiotic and secondary putrefactive changes the entire pulp will finally become totally gangrenous.

When dead protein material is subjected to the action of bacteria and ferments, the process is known as putrefaction. Putrefaction in its early stages is principally a process of hydrolysis and oxidation, and closely resembles tryptic digestion—that is, certain ferments, enzymes and products of bacterial activity are concerned in the cleavage action of the protein molecules, a process which is closely allied to the changes occurring in the intestinal tract. The preliminary action of the proteolytic enzymes on the dead protein molecules results in the formation of albumoses, peptones and polypeptids. Further decomposition is productive of various amino-acids, *i. e.*, fatty or aromatic acids in which one or two of the hydrogen atoms have been replaced by a basic ammonia radical. It is claimed by Czapek and Emmerling that these amino-acids furnish excellent nutritive material for bacteria. The amino-acids are further decomposed by the elimination of ammonia and by the splitting off of carbon dioxid, respectively, deamination and decarboxylization, occurs. In the ammonia elimination the end-products are found to consist of free fatty acids, corresponding to the amino-acids from which they are derived, *i. e.*, acetic, propionic, butyric, valerianic, caproic and a-amino-valerianic acid and of the aromatic acids, *i. e.*, phenyl-propionic, hydro-cumaric, skatol-acetic and succinic acid, etc. The further oxidation of the various fatty and aromatic acids results in the formation of many paraoxy-acid compounds, *i. e.*, paracresol, phenol, etc. Sulphur is set free

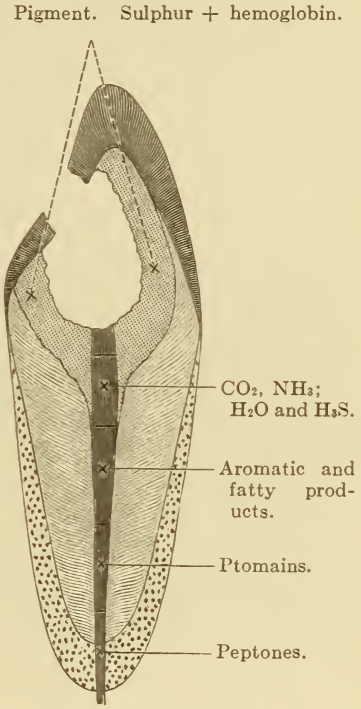


FIG. 80.—Diagram illustrating the more complete decomposition of the pulp at its coronal end. (Burchard and Ingles.)

during the breaking-down of protein substances; partially it unites with free hydrogen to form hydrogen sulphid, and partially with free CN groups to form various sulphocyanids of a less toxic nature. In the course of their decomposition the aromatic products furnish indol and skatol; indol finally combines with free sulphuric acid and forms indican. The latter substance furnishes an important diagnostic indicator of the process of putrefaction.

The products of decarboxylization are amins (tyramin and histamin) and diamins (cadaverin and putrescin) which latter group is included among the so-called ptomains. The aromatic and fatty acids, but especially skatol and indol, are largely responsible for the vile fetid odor which accompanies the putrefaction of protein material. The final products are water, ammonia, hydrogen, hydrogen sulphid and carbon dioxid. This last stage of complete decomposition is rarely reached in the putrefaction of pulp tissue. The reaction of a putrescent pulp is probably always alkaline, and the necessary carbohydrates which would furnish acids as by-products of fermentative changes are absent. The acids that are formed during the decomposition of protein matter are readily neutralized by the many basic materials that are created simultaneously with these compounds. It may be observed, however, that in the union of two amino-acid, an acid radical and a basic radical are liberated, which, under certain conditions, give rise to amphoteric reactions.

The bacterial phase of pulp decomposition is of even greater clinical significance than its chemistry. Hand in hand with the progress of chemical decomposition, the bacteria that are present in the pulp tissue give rise to many substances, *i. e.*, toxins, endotoxins, bacterial proteins and indirectly ptomains.

Mayrhofer[1] furnishes the following statistics concerning the presence of microörganisms in dead pulp tissue:

Organisms found.	Number of times found.
Streptococci	70
Streptococci and rods	44
Streptococci and staphylococci	14
Streptococci, staphylococci and rods	10
Streptococci and yeast cells	5
Streptococci, rods and yeast cells	3
Staphylococci	3
Staphylococci and rods	1
Rods	2

Concerning the presence of these various microörganisms in open and closed putrescent root canals. Mayrhofer obtained the following data:

[1] Principien der Pulpagangrän, 1909.

Organisms found.	Number of times found in 53 cases of open root canals.	Number of times found in 51 cases of closed root canals.
Streptococci	27	31
Staphylococci and rods	18	6
Streptococci and staphylococci	3	1
Streptococci, staphylococci, rods	1	5
Streptococci and yeast cells	1	
Staphylococci	1	2
Staphylococci and rods	1	
Rods	1	6

The influence of bacteria, *per se,* is of little importance as far as pathogenic disturbances are concerned, and the harm that is caused by the presence of these organisms is due to the many chemical products that result in one way or another from their metabolic processes. The many offensive products that accompany putrefactive changes are attributed to anaërobic conditions, while in the presence of oxygen usually less ill-smelling compounds are formed. Some observers claim that only strictly anaërobic bacteria are concerned in the putrefaction of proteins. The autolysis of leukocytes depends principally upon obligate anaërobic microörganisms. Suppurative pulpitis and gangrene, according to Idman, are primarily dependent upon obligate anaërobic bacteria and they play a most important role in these processes. The streptococci and the staphylococci are both aërobic organisms, and only optionally anaërobic. The presence of malodorous compounds is readily perceived by entering into a closed root canal containing a putrescent pulp.

The poisonous chemical products of bacteria, according to Wells, may be conveniently divided into ptomains, toxins, endotoxins and bacterial proteins. The ptomains, *i. e.*, soluble basic nitrogenous substances, resemble vegetable alkaloids and are derived from protein material decomposed under the influence of enzymes and of putrefactive bacteria. For some time past it was believed that ptomains were the cause of infectious disease, but it was soon found that they could be removed from cultures of pathogenic bacteria without destroying the poisonous nature of the latter. At present the chemistry of bacterial intoxication is more clearly worked out, and, as a consequence, ptomains are of much less interest than they were twenty years ago. In decomposing protein material quite a large number of ptomains are more or less present as a result of the cleavage action of enzymes and other hydrolytic agencies. Cadaverin, putrescin, sepsin, muscarin, leucin, tyrosin, neuridin, etc., are some of the more important representatives of this interesting group. Ptomains do not act as specific poisons, but many produce diseases when taken into the body with the food in which they have been produced by bacterial activity. It is claimed that pathogenic bacteria present in living tissue can

not produce sufficient ptomains to seriously affect the health of the individual. Moist gangrene of the pulp is a ready source of ptomain formation.

Certain pathogenic bacteria produce definite synthetic poisonous substances of a specific nature, *i. e.*, the toxins. Toxins are the secretions of cells, and are readily taken up by the surrounding

FIG. 81.—Various types of bacilli isolated from gangrenous dental pulps. (Idman.)

tissues. The intense poisonous nature of these toxins is responsible for the chief symptoms which we recognize in infectious diseases. The bacillus of diphtheria and tetanus are known to secrete typical toxins. These toxins are always of the same poisonous nature, no matter how or where they are obtained, while the ptomains vary with the nature of the substances from which they are derived. Toxins are very labile substances, and they are readily destroyed

by heat, direct sunlight and oxygen. Antibodies or antitoxins can be prepared against toxins, but not against ptomains. As very few bacilli are known that produce specific toxins, it is plain why so few true antitoxins have been artificially prepared.

Again, bacteria may produce poisons within their own cell bodies; they are not usually secreted by the cells, but are also specific in their poisonous nature. These bodies are known as endotoxins. As yet no antitoxins have been prepared against endotoxins, and, as most bacterial diseases are caused by endotoxins, the preparation of sera has been greatly retarded, and consequently, immunization against many infectious diseases is apparently impossible. Furthermore, bacteria contain poisonous

Fig. 82.—Smear obtained from suppurating pulp. Mixed infection; 1 : 1000. (Miller.)

materials which form an integral part of their protein constituents. These poisonous materials are not soluble, and apparently do not produce diseased conditions. The bacterial substances themselves may, however, produce inflammation and pus, or even necrosis, when injected into living tissues. These substances are called bacterial proteins.

Diagnosis.—In complete necrosis or gangrene of the dental pulp when confined to an open root canal usually no painful symptoms are manifested. As soon as disturbances of the periapical tissues occur, which were primarily the sequences of pressure from confined gasses produced by the decomposition of pulp tissue, marked progressively increasing pain with all the additional symptoms of

an acute inflammation of the pericementum are to be observed. The pulp has lost its sensation completely; an explorer may be inserted into the root canal without causing a painful response. Only in partial necrosis or gangrene is pain felt as soon as the line of demarcation is reached. The color of the tooth is markedly altered, *i. e.*, the tooth has lost its life-like luster and it may have changed from the normal to any shade toward dark gray. Transillumination furnishes a dull shadow picture, while temperature changes are not revealed by dead pulp tissue. The percussion sound of a tooth containing a necrosed or gangrenous pulp is negative. The faradic current produces no response whatsoever from a dead pulp; however, great care should be observed in not touching a metallic filling with the electrode or placing the latter too near the gum line, otherwise a painful reaction may be obtained.

If the pulp chamber is exposed the patient usually complains of a bad taste and foul odors arising from the decomposed pulp and from putrescent food particles having lodged in the cavity. Complete loss of sensation and the typical odor of putrefaction are respectively the pathognomonic signs of necrosis and gangrene of the dental pulp.

Prognosis.—Necrosis and gangrene of the pulp indicate dead tissue, consequently treatment cannot be considered. A favorable prognosis of the involved tooth, however, may usually be rendered, as in most instances suitable treatment of the infected root canal and its subsequent filling will restore the tooth to proper usefulness.

Treatment.—The most serious question that confronts the dental profession today—and for that matter has also confronted it in the past—is that which is involved in establishing absolute sterility of an infected root canal. The disposal of this problem in a truly scientific manner necessitates the determination of the established sterility by a bacteriologic or microscopic examination in each individual case. While the writer realizes that the carrying out of such procedures in the average dental office of today will meet with numerous difficulties, due to the fact that the older members of our profession have not had sufficient training in these directions, nevertheless, there exists no valid reason why it should not be done by the recent graduate who has received adequate instructions in laboratory technic, or through a bacteriologic laboratory. The time is not far distant when the public will demand a laboratory diagnosis of serious root canal infections for the same reason that a bacteriologic examination of a diphtheritic throat is demanded at present. Since the sequences of imperfect root-canal sterilization in the form of focal infection resulting in metastatic disturbances of distant organs are of common occurrence, it must follow that the current methods of diagnosis of sterility of a primarily infected root canal are inadequate.

From a logical deduction based upon the above discussed pathology of infected root canals it is evident that its treatment resolves itself into three definite phases: The mechanical, the chemical and the therapeutic procedures. Mechanical manipulations are intended to dispose of the débris of the dead pulp and to assist in the enlargement of the canal. Chemical procedures are primarily applied for the purpose of facilitating the removal of obstructions, and therapeutic applications are utilized to overcome septic condition.

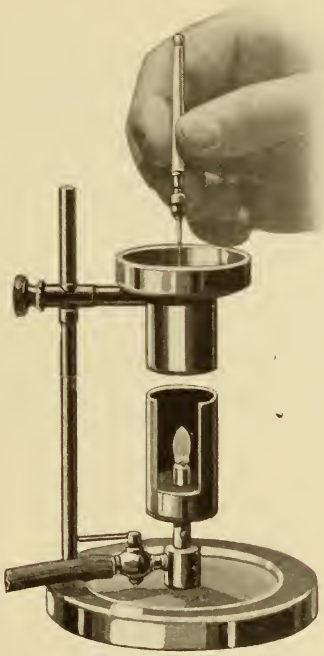


FIG. 83.—The Flaherty molten metal sterilizer.

Sterilization.—It should be an inflexible rule with the operator to perform all root-canal work under strict aseptic conditions. The teeth which are to be placed under rubber dam should be cleaned with cotton pellets and water and the gingival edge is touched with Talbot's iodin solution. The rubber dam is to be thoroughly washed with hot water and soap. The exposed teeth and the adjacent dam is washed with alcohol and dried with bibulous paper. A root canal should only be entered into with absolutely sterile broaches, absorbent paper or cotton points, gutta-percha cones, root canal pluggers, etc. All long-handled instruments should be sterilized by boiling in the usual way in any one

of the ordinary sterilizers, while the delicate root-canal instruments, such as broaches, files, reamers, etc., may be sterilized by dry heat, by using the Flaherty Molten Metal sterilizer or by chemical means as they lose much of their rigidity by boiling in water and, consequently, they readily break. The Flaherty Molten Metal sterilizer is a useful apparatus for sterilizing root-canal instruments, absorbent points, etc. According to laboratory tests as carried out in various institutions, metal broaches, etc., are rendered sterile in five seconds, while paper points and other porous objects require ten seconds when dipped into the molten

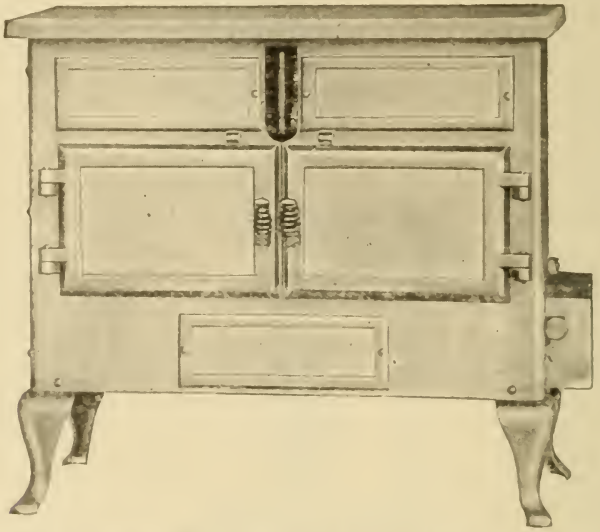


FIG. 84.—Dry heat sterilizer. Automatic control makes it possible to maintain any degree of temperature from 200° F. to 350° F. for an indefinite period. A temperature of 230° F. will sterilize in ten minutes, with a higher degree of temperature less time is required, and with a lower temperature the time must be lengthened proportionately. Especially useful for cotton, absorbent points, gutta-percha cones, napkins, dressings, etc. (Coolidge.)

metal. The temper of the broaches, etc., is not altered by the short exposure to the metal bath. A simple and satisfactory chemical method of sterilization consists in immersing the broaches, files, reamers, etc., in a concentrated (10 per cent) solution of sodium hydroxid for fifteen minutes. This alkaline solution possesses the additional advantage of dissolving organic débris entangled in the barbes of the broaches. A suitable solution is made by dissolving the contents of a can of commercial sodium hydroxid[1] in a gallon of tap water. A heavy precipitate occurs

[1] Crude sodium hydroxid is commercially referred to as "potash" or "lye" and is sold in tin cans containing about 12 ounces of a sufficiently pure preparation for the above purposes.

which is allowed to settle. A glass covered jar of about one-half pint capacity is dipped with its neck into melted paraffin to the extent of about one inch and then filled with the solution just below the paraffin coating. The mechanically cleansed broaches, etc., are placed in a Gooch porcelain crucible with perforated bottom and are immersed in the soda solution. As sodium hydroxid dissolves aluminum, handles made of this metal must be handled with care as it is very caustic. After remaining in the solution for about fifteen minutes, the crucible is removed with

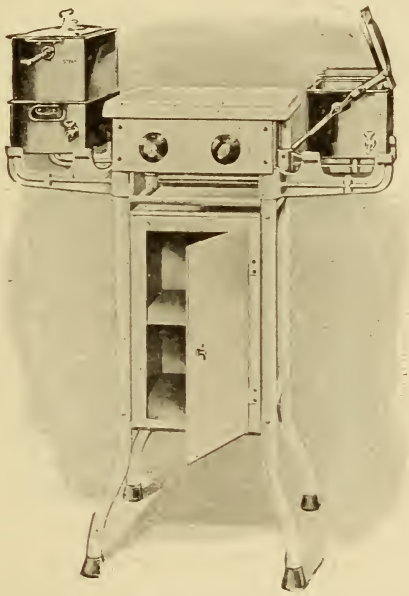


Fig. 85.—Steam chest sterilizer. Moist heat may be obtained varying from 190° F. to 215° F. and can be maintained for an indefinite period. It is necessary to maintain this temperature at least thirty minutes and is safer to sterilize three successive days before using the materials under sterilization. (Coolidge.)

pliers, drained off through the perforated bottom and transferred to a second similar jar (not paraffined) containing a glycerin-alcohol solution, *i. e.*, 1 ounce of glycerin to 7 ounces of alcohol. The root-canal instruments are transferred with sterile pliers to sterile glass dishes (Petri dishes, etc.). The sterilized long-handled root-canal instruments may be readily kept for convenience in suitable bottles. The tips of shears, pliers and other instruments of a similar character and diagnostic wires may be quickly sterilized by dipping in alcohol and burning off in the flame.

A convenient and simple sterilizer for absorbent paper points

and gutta-percha cones has been recently devised by the writer. A colorless screw-cap bottle of about 1 ounce capacity is provided with a piece of thick cotton roll of about one inch in length, which is fastened to the interior of the cap with sealing wax. About 5 drops of dichloramin-T solution (5 per cent) are placed upon the lower end of the roll. The chlorin compound is renewed about once a month. The paper, cotton or gutta-percha points are placed in the bottom of the dry bottle, the screw cap is put into position and the permanent sterilizer is completed. The exposure of the clear glass bottle to the light rays causes a very slow liberation of chlorin. The gutta-percha cones and paper points as obtained in sealed packages from the manufacturers are usually sterile and the little device as suggested is primarily intended to preserve their sterile condition. The efficiency of this simple sterilizer has been repeatedly tested by plating out the points; they have always

FIG. 86.—*a*, Paper-cone sterilizer; *b*, Gutta-percha point sterilizer.

been found to be absolutely sterile. For general office use, a covered glass compartment tray provided with a layer of cottonoid in its glass top upon which a few drops of dichloramin solution are placed in monthly intervals, is very convenient. This dish is known as the "Mynol Compartment Tray." As a most serviceable means of holding the necessary drugs, sterilized broaches, etc., the writer has suggested an aseptic medicament tray. The large compartment of this tray is filled with the glycerin-alcohol mixture and with sterile pliers the necessary instruments are transferred to this compartment and are kept immersed while working on the tooth.

Mechanical Preparation of the Root Canal.—The successful treatment of a root canal depends primarily upon the possibilities of gaining free access to the pulp chamber and to the mouth of the canal. Obstructing walls and angles of enamel and dentin should

be cut sufficiently until free access to all canals on straight lines is secured. It is a good policy to sacrifice a little tooth structure in the beginning which may obstruct a root canal than to try and manipulate broaches around curves and angles. Not too much emphasis can be placed upon this important step of the operation as many of the failures of root-canal treatment are directly traceable to neglect of this initial procedure.

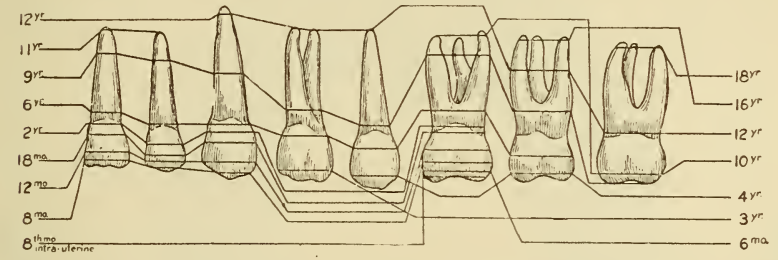


FIG. 87.—Calcification of the permanent upper teeth. (Hopewell-Smith.)

An intimate knowledge of the anatomic structure of the individual teeth, especially in regard to the relationship of their pulps to the surrounding dentinal walls is imperative. One should be mindful of the fact that the dental pulp is the remnant of the original formative organ of dentin. In single-rooted teeth, *i. e.*, incisors, canines and some of the premolars, the pulp occupies the central part of the tooth as regards its long axis, while in multi-rooted teeth with occasional exceptions in the third molars each root contains

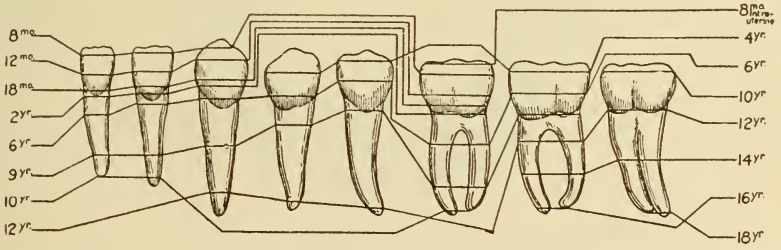


FIG. 88.—Calcification of the permanent lower teeth. (Hopewell-Smith.)

an individual part of the pulp in the same axial relationship, but their pulp chamber is single, *i. e.*, it combines the various root pulps into a single coronal portion.

The size of the pulp depends largely upon the age of the respective individual; with increasing age a diminution of its bulk is to be observed. In the young an intimate knowledge of the stage of calcification of the respective tooth is imperative as the size of the

pulp and of the apical foramen within certain periods of age varies greatly. Up to middle age the volume of the pulp remains fairly constant while in the aged new irregular masses of dentin are

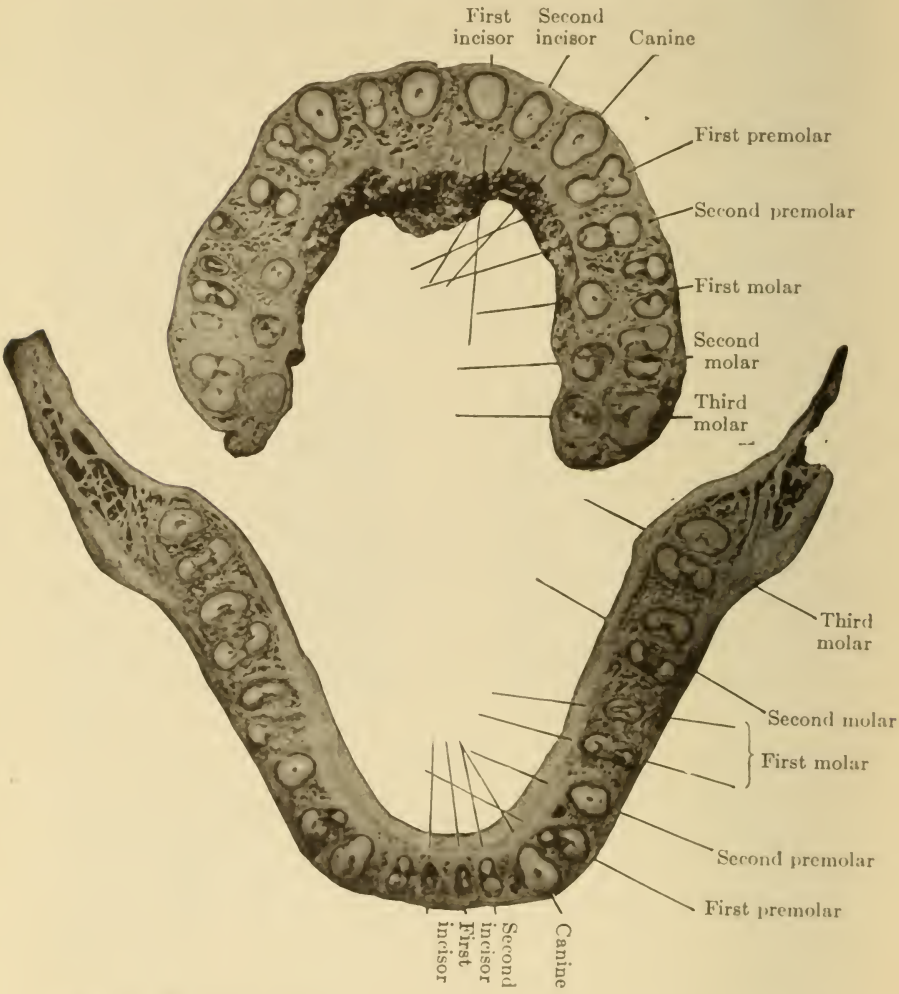


FIG. 89.—Horizontal sections of the maxilla and mandible cut a little beyond the free margin of the alveolar process, showing the forms and positions of the roots of the various teeth. (Cryer.)

deposited within the canal which may be the cause of senile atrophy of the pulp and usually of more or less complete obliteration of the root canals.

In general, it may be stated that the pulps of the incisors and the

canines resemble slightly depressed cones, ending in a thin thread near the foramen, while in the crown they may reach to within a third and occasionally to one-half of that portion of the tooth. In the crown the pulps of the incisors end in three slight eminences, while the coronal part of the pulps of the canines present single sharp points.

The pulps of the upper first premolars present in their coronal portion two distinct horns corresponding to the two cusps; the lingual horn is the smaller of the two. The buccal horn often extends within the center of the crown while the lingual horn rarely passes beyond the neck of the tooth. The bucco-lingual diameter of the pulp is larger than the mesio-distal diameter with a slight depression near the center. The two root canals of this tooth do not always separate within the pulp chamber; the isthmus is often found in the first third of the root or even at its center. The upper second premolar is usually single rooted and consequently only one canal is observed. Frequently, however, a division of the pulp may occur within the upper third of the root. The diameter of the pulp is much wider bucco-lingually than in its mesio-distal direction.

The pulps of the upper molars, especially in young individuals, usually show a pronounced coronal portion corresponding to the three and, rarely, to the four cusps of its crown. The coronal portion of the pulp rarely occupies more than a third of the crown. The pulp canal of the lingual root is the largest of the three canals; occasionally it may be bifurcated near its apical end. Of the two other canals the disto-buccal canal is the smallest and in conformity with the outline of the root it is frequently curved.

In the third upper molars the pulp canals follow the general outline of the very variable number of roots. But as this tooth has a tendency to be most irregular in size and shape the number of root canals differs greatly. Frequently in the single-rooted third molars only one large central canal is observed which may show several bifurcations near the apex.

The pulp of the lower incisors and canines differ little from those present in the same teeth in the upper jaw. In most instances, they reach far up into the crown. Occasionally, a bifurcation of the root canal is to be observed in the lower canines.

The lower premolars are single-rooted teeth and usually they are regular in outline. Frequently the pulp presents two horns, of which the buccal is the largest. The greatest diameter is observed near the neck of the tooth in a bucco-lingual direction. Near the apical end the pulp often tapers to a fine thread.

The coronal portion of the pulps of the lower molars corresponds to the general outline of the crowns of these teeth; usually four horns are observed. The pulp canal of the mesial root frequently

divides into a buccal and a lingual portion while the distal root presents in most instances a single well-developed canal. The greatest diameter of these canals lies in a bucco-lingual direction.

FIG. 90.—Diagram of longitudinal sections of permanent teeth showing the spaces which the pulps occupied.

The pulp canals of the lower third molars are very irregular in outline; in single-rooted teeth a large pulp cavity with a single or several canals may be observed. In the latter instance these canals frequently ramify.

Many anomalies of the normal topographic anatomy of the dental

pulp may be observed. Among the principal malformations may be mentioned numerous branches of the pulp which are directly corrected through lateral canals with the pericementum. These ramifications are especially numerous near the principal foramen; five or even more such branches have been observed. Connecting bridges within the root canal which contain pulp tissue are often encountered. Additional roots with root canals and, consequently, branches of the pulp, are by no means rare abnormalities; usually they may be diagnosed from a good roentgenogram.

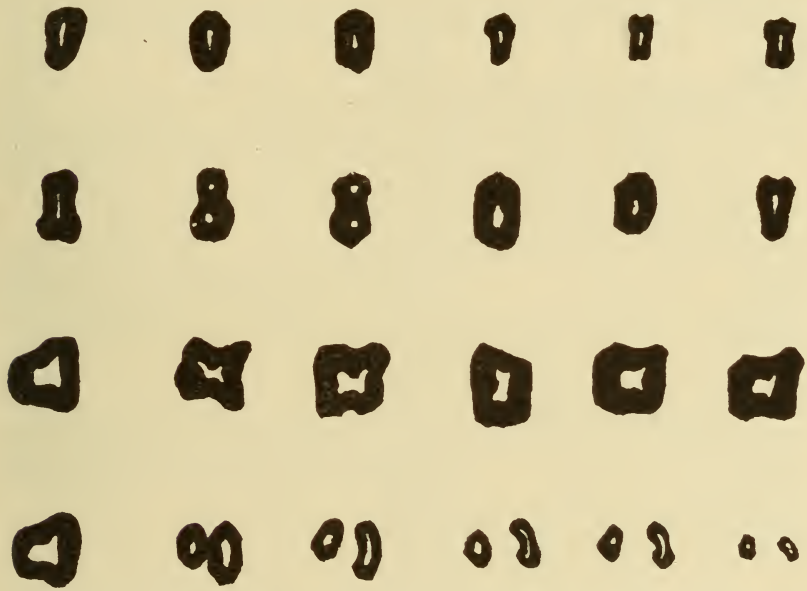


FIG. 91.—Diagram of cross sections of permanent teeth at various points.

The successful removal of the débris from a root canal depends primarily upon its size; large canals are naturally more accessible than the small, tortuous canals, especially those of the mesial roots of the lower molars, the buccal roots of the upper molars and those of the upper first premolars. Curvatures of the roots and the presence of secondary dentin materially increase the difficulties. During the process of enlarging the canals numerous complications may arise, among which the packing of débris into the apical regions of the very fine canals, cutting ledges into the walls of a curved canal and breaking the tip of a broach near a curve are relatively common occurrences. At the beginning of the operation only the very finest picks or pathfinders should be employed which may be followed with barbed broaches. Binding of the broach

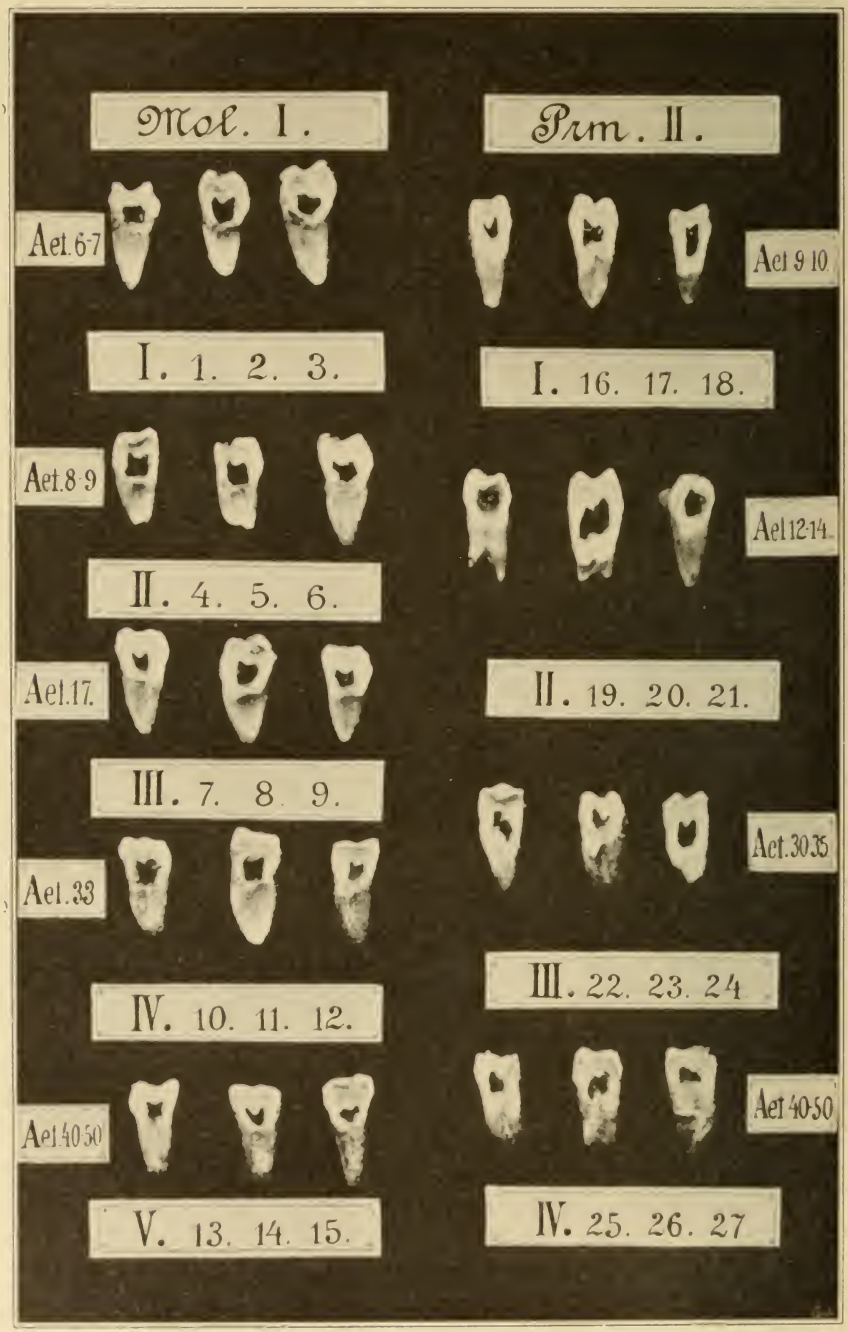


FIG. 92. — The size of the pulp chambers of permanent teeth at various ages. (Szabo.)

within the canal must be rigidly avoided as breaking of this delicate instrument by applying undue force is almost certain to occur. If a ledge is cut, which is usually the result of using too large a broach,

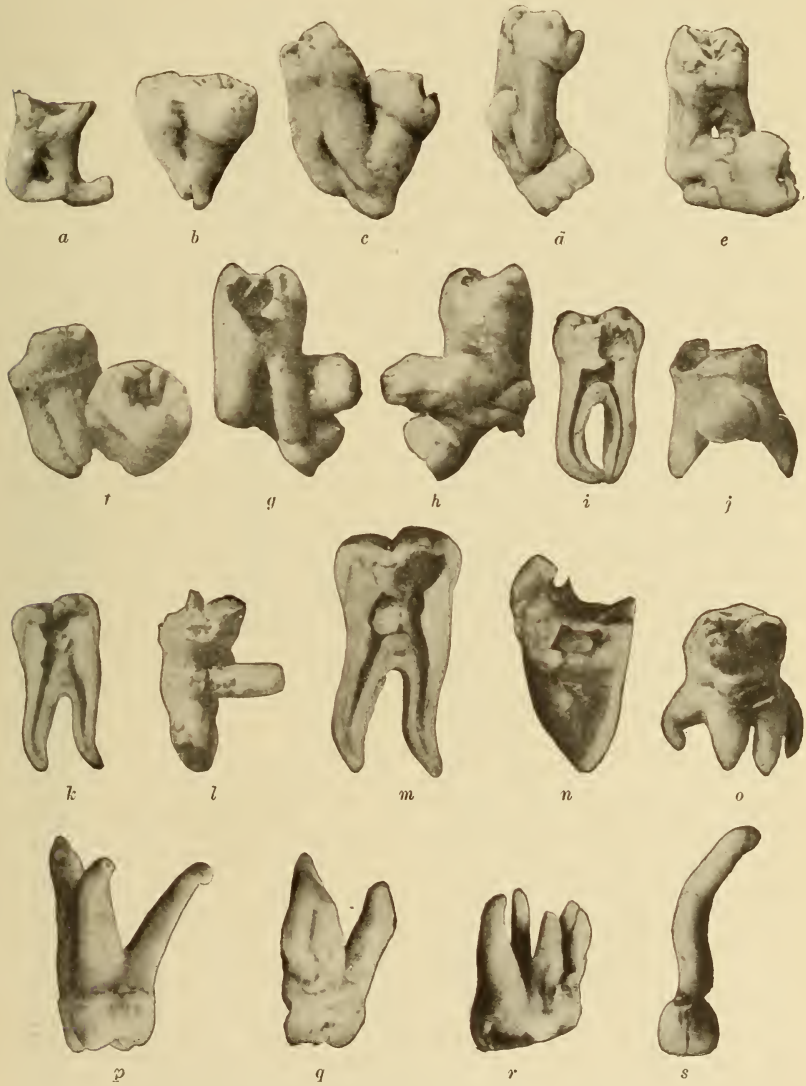


FIG. 93.—A group of abnormal teeth. (Cryer.)

it must be carefully filed away; otherwise the formation of a pocket and finally a perforation of the wall of the root is very apt to take place. By alternating the various sized picks and broaches

the canal is finally cleansed of its débris and the apical foramen is brought within reach. Very delicate apex curettes, the so-called

Fig. 94.—Pulp canal cleaners.

apexographers, are now employed for enlarging the lumen of the apex by drawing the débris into the canal. With suitable Kerr files the canal may now be enlarged and straightened so as to assume a conical shape which may materially assist in placing the gutta-percha cone simultaneously against the wall and the apex.

Fig. 95.—Donaldson pulp canal cleaner.

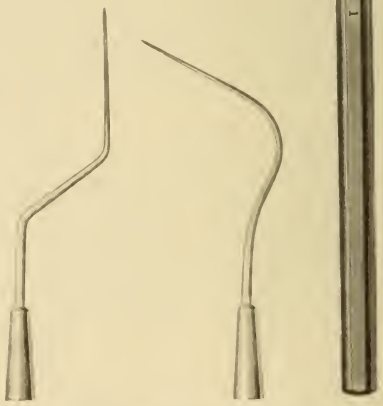


Fig. 96.—Rhein's picks.

Consultation of the roentgenogram with the diagnostic wire in position should be frequently resorted to. As stated, special attention must be given to the upper third of the canal, including the foramen. This space is the most important part of the root canal in regard to the future health of the periapical tissues, which is largely dependent on the successful occlusion of this region by the root-canal filling.

Chemical Procedures.— For the chemical disintegration of the pulp detritus, but primarily for the purpose of assisting in the opening of

obliterated root canals, two specific methods are in vogue, *i.e.*, the alkali method, as introduced by Schreier[1] in 1892, and the acid method, as advocated by the late John Callahan[2] in 1893, Schreier's alkali method intends to destroy the organic constituents of the calcareous deposits and, incidentally, to assist in the dissolution of necrosed tissue by means of the freshly formed hydroxids of potassium and sodium derived from an alloy of potassium and sodium in the presence of water and thereby rendering the remaining inorganic débris more friable and offering less resistance to the advancing instruments, while Callahan's sulphuric acid treatment produces the opposite effect, *i. e.*, it destroys the inorganic substances by dissolution and carbonizes the remaining organic material. Both methods have their advocates and they virtually accomplish the same purpose. Preference in regard to selecting either method is largely a matter of personal equation. However, a most important physical property possessed by the alloy and

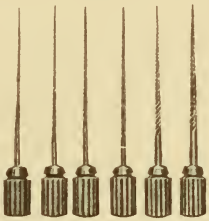


Fig. 97.—Root canal files.

which is not possessed by the acid, should be mentioned which places the alkali method at a marked advantage over the acid treatment. This property manifests itself as a pronounced capillary affinity of the freshly formed hydroxids for moisture and, consequently, the caustic alkali solution penetrates into those minute apertures of the root canal in which the acid will *not* flow. This fact combined with the solvent action of the alkalies on necrosed tissue is of the utmost significance in regard to the opening of those exceedingly narrow canals which are often too small to admit the finest broach. Both methods incidentally destroy the offensive odor of putrescence which is always very pronounced in a closed root canal and less so in an open one. However, it should be clearly understood that the mere absence of foul odors is by no means a criterion of sterility.

[1] World's Columbian Dental Congress, 1893.
[2] Proceedings of the Ohio State Dental Society, 1894.

Dental potassium-sodium (kalium-natrium) usually consists of 1 part of metallic potassium and 2 parts of metallic sodium melted together beneath kerosene. It is of a pasty consistency and resembles mercury in appearance. The alloy is applied in small glass tubes sealed with wax or paraffin for protection. The tube must be hermetically sealed directly after using the alloy to prevent its decomposition by absorbing moisture from the air. If the sealing is not carried out in a proper manner the operator will find that the contents of the tube will change to a hard, crystalline mass, *i. e.*, the hydroxids of the two metals.

Fig. 98.—Tube of sodium-potassium alloy.

Fig. 99.—Iridio-platinum nerve broach. This broach is not corroded by sodium-potassium alloy and may be sterilized by heating to redness.

In using this alloy, a barbed tantalum or an iridio-platinum broach is thrust through the paraffin stopper or directly into the broken off upper end of the tube. Steel broaches are not to be recommended for this work; the alloy disintegrates the metal and, as a consequence, the broaches frequently break in the canal. The very small quantity of the paste adhering to the broach is worked into the pulp débris. Extreme care should be exercised in regard to the quantity applied to the canal. Only the very thin film, free from lumps, which will adhere to the broach is permissible to be used with safety. At once a chemical decomposition of the

moist contents of the root canal takes place manifesting itself by heat and a hissing sound with the escape of gas, and, if larger particles are used, with little sparks of fire. Potassium-sodium alloy in the presence of water is changed at once into their hydroxids with the liberation of hydrogen. The hydroxids dissolve in the remaining water and form a more or less concentrated caustic alkali solution. The putrescent pulp, as stated above, contains water, fat, fatty acids, gases and the débris of protein material. The rationale of the action of the potassium-sodium alloy on the putrescent pulp débris may be summarized as follows: Fat and fatty acids are changed to soluble soaps. The protein substances are rendered soluble by the caustic hydroxid solution and the liberated hydrogen forces the undissolved débris to the surface of the canal. The calcareous deposits in the lumen and upon the walls of the canals lose their orderly structure and become friable and thereby offer less resistance to the advancing broach. The offensive odor of putrescence is almost instantly destroyed. Copious washing with water will remove the saponified contents of the canal and on drying, its clean, ivory-white walls are visible. The substitution of the hydroxids of potassium or sodium for the metallic alloy as suggested by Schreiter is not to be recommended; their application is difficult and their physical nature does not lend itself to this procedure as readily as the alloy from which the above hydroxids are obtained in the nascent state during their application. Alcoholic solution of sodium ethylate or methylate in various concentrations, *i. e.*, from 2 to 10 per cent has also been recommended for the same purpose.

Sodium dioxid, as suggested by Kirk in 1893, when applied in substance to the moist contents of the root canal, is a most serviceable chemical adjunct for the ready disintegration of the necrosed tissue. It is carried into the canal by means of a broach previously dipped into chloroform, absolute alcohol, glycerin-alcohol mixture (1 to 7), etc. These liquids merely act as indifferent conveyors of the salt, which, if water or a low grade alcohol were used as a vehicle, readily decompose the sodium dioxid. The decomposition of the latter into nascent oxygen and sodium hydroxid exercises a most beneficial and multifarious effect upon the necrosed pulp, which in many respects, is equal to that obtained from potassium-sodium alloy.

The sulphuric acid method for opening obstructed root canals has found many admirers among the practitioners and it is probably more widely employed at present than any other chemical procedure. Other acids, such as hydrochloric, nitro-hydrochloric and phenol-sulphonic acid have been advocated for this purpose during the last twenty-five years. The strength of these various acids as used for this specific purpose should be carefully noted as

it varies greatly. Sulphuric acid is usually employed in approximately a 50 per cent solution, hydrochloric acid in a 10 to 30 per cent solution, nitro-hydrochloric acid in a 50 per cent solution, while phenol-sulphonic acid is used in its pure form only. The therapeutic absurdity of the latter compound has been dealt with by the writer on a former occasion[1] and consequently it is omitted in the present discussion. The substitution of phenol-sulphonic acid for this or any other purpose in which sulphuric acid is indicated in dental therapeutics means the application of a decidedly inferior remedy.

Nitro-hydrochloric acid (aqua regia) as introduced by G. W. Weld[2] in 1897, is a very efficient acid for the above purposes. On account of its strong caustic properties, extreme care is required in handling it. The acid should be preserved in glass-stoppered bottles and kept outside of the operating room, as its fumes are most destructive to metallic instruments. The greatest solvent power upon dentin is possessed by so-called "reversed" aqua regia, *i. e.*, an acid composed of 1 part of hydrochloric acid and 4 parts of nitric acid. This acid should be applied upon an iridio-platinum or a tantalum broach wound with a few fibers of asbestos to facilitate the mechanical adhesion of the acid. Steel broaches are most readily destroyed by the corrosive action of this acid.

To facilitate the ready application of these acids to the root canals it has been suggested to prepare a suitable paste by adding insoluble barium sulphate to the acid. The writer cannot advocate such mixtures.

Whatever acid is employed it should be neutralized by sodium dioxid conveyed to the canal as described above and *not* by sodium bicarbonate, which is practically of very little value for this specific purpose. If sodium dioxid is used in connection with an acid it should preferably be applied first and then neutralized by the acid. The chemical interchanges between the various acids and the sodium dioxid may be portrayed by their respective reactions as follows:

$$H_2SO_4 + Na_2O_2 = Na_2SO_4 + H_2O_2$$

or

$$2HCl + Na_2O_2 = 2NaCl + H_2O_2$$

or

$$2HNO_3 + Na_2O_2 = 2NaNO_3 + H_2O_2$$

and $$2HCl + Na_2O_2 = 2NaCl + H_2O_2$$

The freshly formed hydrogen dioxid of the last reaction combines with the available hydrochloric acid and forms nascent chlorin:

$$H_2O_2 + 2HCl = 2H_2O + Cl_2$$

[1] Prinz: Dental Cosmos, 1912, p. 397.
[2] Dental Cosmos, 1897, p. 397.

There seems to be quite a divergency of opinion concerning the self-limiting action of these acids upon tooth structure. Fifty per cent sulphuric acid solution is self-limiting; a tooth placed in this acid will be coated within a day or so on every accessible surface with freshly precipitated insoluble calcium sulphate and consequently no further action occurs. A tooth placed in 10 per cent hydrochloric acid, in pure or in 50 per cent nitro-hydrochloric acid will be completely dissolved in two or three days; these acids form soluble calcium salts. The greatest solvent power is exhibited by the "reversed" nitro-hydrochloric acid. The various reactions of these acids with one of the constituents of the dentin, calcium carbonate, may be depicted as follows:

$$H_2SO_4 + CaCO_3 = CaSO_4 + CO_2 + H_2O$$
$$2HCl + CaCO_3 = CaCl_2 + CO_2 + H_2O$$
$$2HNO_3 + CaCO_3 = Ca(NO_3) + CO_2 + H_2O$$

From a clinical point of view it may be readily observed that the small quantities of either acid pumped into a root canal, when used with caution, will do no harm, especially when neutralized by sodium dioxid. It is to be understood, however, that no acid should be forced through the foramen. This same precaution is equally true in regard to the use of potassium-sodium alloy, sodium dioxid, etc.

Incidentally, it should be observed that these various chemical agents by their intense caustic effects naturally also act as powerful germicides. Sulphuric acid is the weakest while hydrochloric acid, and especially nitro-hydrochloric acid, when neutralized with sodium dioxid develops a marked degree of disinfecting action on account of the liberated chlorin. Potassium-sodium alloy is a most powerful germicidal agent; the freshly formed hydroxids dissolve every vestige of organic material with which they come in contact. The intense penetrating power of these hydroxids is of material assistance in reaching the minutest ramifications of the root canal. As a consequence, quite a few practitioners depend exclusively upon the chemical action of potassium-sodium alloy in rendering the surface of a root canal sterile.

Nascent oxygen which is created by the reaction of sodium dioxid with any of the acids employed and nascent chlorin obtained from the nitro-hydrochloric acid and sodium dioxid reaction as depicted above are important additional chemical factors evolved in the treatment of the root canal. These bleaching agents will materially assist in the preservation of the natural color of the tooth. Copious washing of the canal with water after the chemical treatment should always be employed to get rid of the products of the various reactions and of the débris. The most satisfactory results from the utilization of the above discussed chemical pro-

cedures for chemical purposes are obtained by the use of potassium-sodium alloy or by the combined application of the alkaline and acid methods in logical sequence, *i. e.*, sodium dioxid followed by sulphuric or nitro-hydrochloric acid and finally, by copious washing with water.

Therapeutic Procedures.—Prior to the introduction of the antiseptic era by Lister in 1867, ancient and confused delusions of miasm, contagion and virus had taken possession of the mind of the dental practitioner. He looked for suppuration of the dead "nerve" in the afflicted "fang" and if the oozing pus was not *bonum et laudabile*, he would waste little time in the treatment of the offending tooth; he usually removed it. If a suppurating pulp was present in an anterior tooth and he wished to save the latter he would drill a small hole in the pulp chamber at a convenient place somewhere near the free margin of the gum to give ready vent to the accumulated accretions. Archigenes (about 130 A.D.) had introduced this operation and devised a small trephine for the purpose. This same procedure was advocated as a new venture by Hullihen in the middle of the last century.

Soon after the inauguration of the antiseptic era in surgery, in 1867 by Lister, dentistry adopted his methods for the treatment of root canals in an empiric way by using phenol as advocated by Witzel in 1873. Since then innumerable other drugs and drug compounds have been recommended at various times for this purpose among which may be mentioned: Creosote, chloro-phenol, camphorated phenol, lysol, cresol, creolin, betanaphthol, salicylic acid, hydrogen dioxid, zinc chlorid, mercury bichlorid, silver nitrate, iodin solutions, iodoform, the essential oils, thymol, eugenol, eucalyptol, Black's 1–2–3, sodium dioxid, formaldehyd, electro-sterilization (ionization), dichloramin-T and many others too numerous to mention. From a clinical point of view the cresol-formalin mixture as introduced by Gysi in 1899, and which was widely popularized by Buckley in 1904, has received greater approval than any other medicinal compound recommended for such purposes. The true criterion of an antiseptic must be based upon its bacteriologic efficiency as tested in clinical cases. The high standard of the germicidal activity of formalin has been frequently established by rigorous experiments. Clinical data collected in the early days of the use of the above mixture pointed to most favorable results. In due time, however, it was observed that while "clearing up" of an infected root canal, as far as the ordinary diagnostic evidence is concerned as applied in the average dental office, *i. e.*, absence of foul odors, occurred much more rapidly by the use of this mixture than by employing any of the numerous other drugs usually advocated for this purpose, nevertheless, secondary manifestations about the periapical tissues were of fre-

quent occurrence. These disturbances are an indication that the supposed sterility of the canal was not obtained at the time of its treatment with the cresol-formalin mixture, or that this compound produces a predisposition of the periapical tissues to future infections. To be sure, dental literature is replete with statements such as this (referring to the cresol-formalin mixture): "This dressing should remain for at least three days, by which time the remedy will have sterilized the entire tubular structure of the dentin, thus establishing asepsis." As no bacteriologic proof is furnished to substantiate this empiric statement it does not carry any scientific weight, and it is out of harmony with existing facts. Asepsis of the surface of an infected root canal can be temporarily established by applying mechanical and chemical measures, but complete sterilization of "the entire tubular structure of dentin" in a tooth *in situ* is impossible with the methods at present in vogue.

In regard to the application of powerful antiseptic drugs for the treatment of infected root canals, one should always be mindful of the following facts:

1. The agent must be able to develop the highest degree of antiseptic power without doing harm to the periapical tissues.
2. It must maintain its activity for at least twenty-four hours when sealed into the root canal.
3. It must not cause pain.
4. It must not discolor the tooth structure.

Of all the above enumerated drugs only a very few answer these requirements. Without entering into a lengthy discussion of the merits or demerits of these drugs, it may be stated that, in general, the antiseptics of the aromatic series, *i. e.*, phenol and its isomers act as caustics when applied in concentrated solution. The metallic salts are strong precipitants of albumen; incidentally, some of these salts, *i. e.*, mercury bichlorid, silver nitrate, etc., permanently discolor the tooth structure. Iodoform, on account of its most disagreeable odor and other drawbacks cannot be recommended for this work and some of the iodin compounds produce lasting stains. The essential oils do not possess sufficient antiseptic power as compared with other drugs. Formaldehyd in the strength in which it is usually applied for root-canal treatment will always kill soft tissues when brought in contact therewith either directly or in vapor form in the same manner as the ill-fated desensitizing paste by its formalin content eventually kills the pulp through any thickness of sound dentin. The clinical practitioner should be mindful of the following dogmatic axiom: Never seal into a root canal a drug which by its passage beyond the apical foramen may cause cauterization of the soft tissues. Regarding the action of antiseptics as employed in the treatment of infected root canals,

practitioners are rather prone to depend too much on the specific activity of such compounds. There seems to exist a current conception that neglect in regard to the mechanical removal of infected pulp débris may be overcome by the application of powerful germicidal drugs. This inaccurate impression is partially the outcome of misinterpreting results as observed in test-tube experiments and partially it is based upon exaggerated claims made by manufacturers of certain antiseptic compounds. There can be nothing more erroneous than such perverted conceptions regarding drug action. Neglect of the most painstaking mechanical removal of pulp débris and chemical cleansing of the canal can never be counter-balanced by the future application of drugs. As will be pointed out in detail later under "Reinfection of Root Canals," no antiseptic treatment now in vogue will permanently sterilize the once infected contents of the dentinal tubuli.

Chlorin as an Antiseptic.—Of all known antiseptics, chlorin, freshly prepared, and in the presence of moisture and in a suitable concentration, especially in an oily solution, is harmless to the periapical tissues and it maintains its activity for about twenty-four hours when sealed into a root canal. These oily chlorin solutions will not cause pain and they do not discolor but rather bleach tooth structure.

From an historical point of view it is interesting to observe that chlorin in the form of its numerous preparations has had a rather checkered career within that group of substances generally referred to as disinfectants and antiseptics. In 1788 the French chemist Berthollet obtained a liquid which exhibited marked bleaching and disinfecting properties, and Tennant, of Glasgow, in 1798, prepared a more stable compound in the form of chlorinated lime for similar purposes. In 1792 the Javelle works, near Paris, prepared a liquid bleaching compound for commercial purposes by passing chlorin into a potash solution, which since has become widely known as eau de Javelle or Javelle water. Again, in 1820, Labarraque, a French pharmacist, modified this latter solution by substituting sodium carbonate for the potash salt. This solution, variously known as Liqueur de Labarraque, as Labarraque's disinfectant fluid, or at present simply as Labarraque's solution, achieved great renown at the death of Louis XVIII, "for thanks to the disinfecting and deodorizing value of his liqueur Labarraque was able to proceed with the embalming of the royal body, which was so profoundly decomposed that no one was able to approach it until after the application of the hypochlorite." (Dakin.)

When Semmelweiss, the assistant obstetrician of the Vienna Lying-in Hospital, announced, in 1846, his epoch-making observation concerning the clinical causes of puerperal fever, he selected chlorinated lime solution as the agent par excellence for the destruc-

tion of that loathsome unknown "miasm" which was carried by the students, as he observed it, from the dissecting room to his wards, and which was the apparent cause of the scourge. Again, during the civil war, chlorinated lime solutions were occasionally employed to check the frightful destruction of human life by hospital gangrene. However, principally on account of the caustic effect of the solution employed, together with a faulty technic in its application, the compound never obtained the general usefulness which it rightly deserved. With the advent of modern bacteriology, chlorin has always been a favorite antiseptic in the hands of the experimental researcher, and within the province of the hygienist its application for the purification of drinking water and the sterilization of excreta it has achieved most remarkable results. and consequently it is at present very largely employed for such purposes.

Repeated attempts have been made in the past by bacteriologists to convince clinical surgeons and dentists that chlorin solutions are the acme of perfection as far as wound antiseptics are concerned. As a consequence, various preparations under more or less fanciful names have appeared on the market, among which Labarraque's solution, Javelle water, electrozone, dental meditrina, radizin, antiformin, eusol, eupad, chlorazene and many others are examples. All of these solutions show remarkable activities as far as their laboratory tests are concerned; nevertheless in their practical application they do not produce the same satisfactory results, and hence they have proved disappointing to the clinician. It should be borne in mind that most of these preparations are strong irritants, and that chlorin solutions are rather labile compounds; all of the solutions named loose their activity within a week or two, hence the disappointing results when commercial stock preparations are employed.

Pharmacology of Dichloramin-T.—The halogens represent the most interesting and incidentally the most important group of those chemical substances which as a class are referred to as wound antiseptics and as general disinfectants. On account of their general behavior bromin and fluorin are of less importance for such purposes, while iodin and chlorin, because of their high efficiency and their universal adaptability from a clinical point of view, form the most important group among these substances.

At this moment chlorin is considered "king" among the chemical disinfectants; when freshly prepared, in the presence of moisture and a suitable temperature, it is the most active disinfectant known. From a technical point of view, however, numerous difficulties arise in its application which in the past have more or less interfered with its universal adaptation. Chlorin as such is a gas which, while it may be readily compressed into a liquid, cannot be employed

with any degree of satisfaction for wound treatment, as it is too irritating. Suitable solutions prepared from the liquefied gas, a process which at present is very much simplified, possess necessarily all the drawbacks of an aqueous solution.

The usual solutions which have been most widely employed are Labarraque's solution, *i. e.*, a solution of chlorinated soda and Javelle water. These two solutions, however, possess also serious drawbacks as far as their clinical application is concerned. They readily deteriorate and, incidentally, they macerate the epidermis when kept in contact therewith even for a short time only, so as to render them practically not only useless but directly dangerous for the purpose for which they are intended.

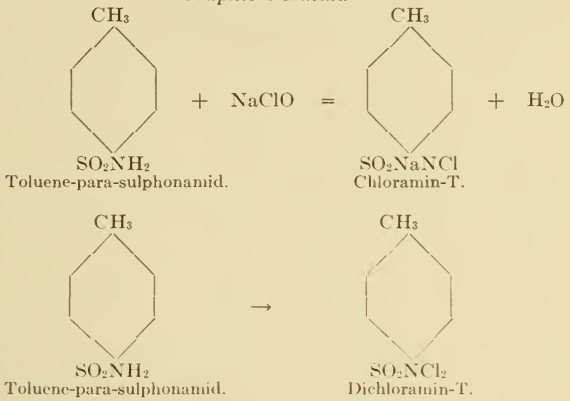
Dakin, in his studies of the antiseptic properties of the hypochlorite of soda solution, finally succeeded in preparing a compound which is practically neutral and which incidentally contains only about 0.5 per cent of the hypochlorite salt. This solution produces virtually no irritating action on the skin and wound surfaces, and it is the compound which at present, according to the Carrel-Dehelly-Depage technic, is employed in the treatment of wounds. The great success obtained with Dakin's solution rests primarily upon the fact that a fresh preparation according to specific methods is employed, which, while acting deleteriously on the germs, does little harm to the tissue cells, as it is a non-irritating isotonic wound antiseptic. The rationale of sterilizing an infected wound surface by the Carrel method is based on the following conception: "To render an infected wound sterile it is necessary to employ a suitable antiseptic in such a manner that the chosen antiseptic comes into contact with every portion of the wound, that the antiseptic is maintained in a suitable concentration throughout the entire wound, and that this constant strength is maintained for a prolonged period. If these conditions are fulfilled every wound will show its response to the treatment by the diminution and disappearance of its micro-organisms." Without entering further into a discussion of the complicated and time-consuming technic of the above method, it suffices to say that the method as well as the solution employed is practically useless for the treatment of infected root canals.

Dakin soon realized the clinical drawbacks possessed by the various solution of chlorinated soda, and his further studies led him to utilize certain synthetic chlorin compounds discovered by Chattaway, among which chloramin-T and dichloramin-T are the two most important representatives. Chloramin-T, being water-soluble, possesses more or less the same disadvantages as any other aqueous solution of chlorin. The permanence of such solutions is very limited, and, further, they are very quickly exhausted by wound secretions. On the other hand, dichloramin-T, a substance soluble in an oily medium, furnishes a veritable reservoir

for the slow elimination of chlorin which preserves its activity for many hours in the presence of wound exudates.

Dichloramin-T is the abbreviated name of para-tuluene-sulphone-dichloramid, $CH_3C_6H_4SO_2NCl_2$. (The letter T, indicating toluene, distinguishes it from similar compounds which have been or may be made from benzene, xylene, etc.)

Graphic Formula



It is a yellowish-white crystalline powder, having a sweetish, rather pungent chlorous odor and containing a little over 29 per cent of available chlorin. It melts at about 80° C. (176° F.). In the solid state, when kept in the dark it is stable. It is practically insoluble in water, but is readily soluble in most organic solvents, *i. e.*, chloroform, benzene, eucalyptol, etc. It quickly reacts, undergoing decomposition with the evolution of nascent chlorin when brought into contact with most organic substances, such as acids, alcohol and the amins, with hydrogen dioxid, water, etc., and certain metals. It should be stored in small amber-colored glass-stoppered bottles and protected from heat.

The strength of dichloramin-T or its solutions may be readily estimated by taking an aliquot quantity, *i. e.*, 0.1 gm. of dry dichloramin-T or 0.5 gm. of its solution and adding 5 cc of carbon tetrachlorid and an excess of a 10 per cent potassium iodid solution and glacial acetic acid. The liberated iodin is titrated with $\frac{N}{10}$ sodium thiosulphate solution. Each cubic centimeter of the thiosulphate solution is equal to 6 mg. of dichloramin-T. A strong odor of chlorin and incomplete solubility in chloroform are reliable signs of decomposition of the salt, while extreme turbidity and the formation of crystals in the bottom of the bottle are indications of the decomposition of its solution in chlorcosane.

Solvents for Dichloramin-T.—At the early stages of our experimental work we prepared the dichloramin-T solution in accordance

with the original suggestion of Dakin, by using such solvents as chlorinated eucalyptol and chlorinated paraffin oil, or a mixture of both. However, it was observed that these solutions produced varying degrees of pain, which we attribute to the irritating effects of the solvents. Chlorinated eucalyptol is an unstable body which readily decomposes in the presence of dichloramin-T by absorbing moisture, resulting in the production of volatile acid substances. Hence when Dakin announced that he had prepared a new solvent for his antiseptic which eliminated the above disadvantages of the original solvents, we at once tried out the preparation and found it to be fully in accordance with the claims made for it. This new compound is known as "chlorcosane." It is a bland, heavy, viscid oil, having a slight yellowish color, and is prepared from hard paraffin melting at about 50° C. (122° F.) by replacing a part of its hydrogen by chlorin. Chlorcosane does not contain any "free" chlorin, although it absorbs from 45 to 55 per cent of its own weight. The chlorin combines with the carbon of the paraffin somewhat in the same manner as chlorin and sodium combine to form the ordinary inert sodium chlorid. Chlorcosane, by the application of moderate heat, will readily dissolve from 8 to 10 per cent of dichloramin-T, which is more than amply sufficient for dental purposes. As the preparation is too cumbersome to be attempted by the dentist, chlorcosane, as well as dichloramin-T is best procured through the ordinary trade channels.

Preparation of Dichloramin-T Solution.—Regarding the concentration of the solution of dichloramin-T for the purpose of treating infected root canals, we have found that a 5 per cent solution of the salt in chlorinated paraffin, *i. e.*, chlorcosane, answers our purpose quite satisfactorily. We have heard an opinion expressed to the effect that a 5 per cent solution is too irritating when used in root-canal work. We cannot subscribe to such assertions; we rather believe that the pain resulting from its application is usually due to two causes—a spoiled solution and a faulty technic.

Solutions of dichloramin-T preserve their activity for a limited time only; they usually deteriorate within a month or so and, therefore, it is best to prepare a convenient quantity which may be readily used up within a few weeks. To prepare a dram of the solution, which will be amply sufficient for a week's supply for the average dental office, 3 grains of dichloramin-T are placed in a test-tube which must be absolutely clean and free from moisture. A few drops of chloroform are added, the tube is shaken and 1 dram of chlorocosane is now added and the whole is thoroughly agitated. The tube is plugged with cotton and placed upright in a sand bath and heated. Within a quarter of an hour complete solution usually results. The heat of the sand bath must never be above 194° F (90° C.). Overheating must be carefully avoided

as it will decompose the solution almost immediately. The solution is now ready for use; filtering is not necessary. As stated above, only dark amber-colored or black bottles should be employed as storage vessels; blue glass does not protect the solution against the actinic effects of strong light.

Solution of dichloramin-T must be carefully protected against heat, light, water, alcohol and most metals; in fact, most common substances have a strong affinity for chlorin, hence the ready decomposition of this solution when brought in contact therewith. Whenever the solution becomes turbid and forms a deposit of crystals in the bottom of the bottle or develops a pronounced odor of hypochlorous acid it should be discarded. Fresh solutions, if chilled, may temporarily become cloudy, or even precipitate, owing to the separation of either dichloramin-T or of solid paraffin. Slightly warming the solution quickly restores its usefulness.

FIG. 100.—Office-preparation bottle.

For office purposes it is best to keep the dichloramin-T solution in an amber-colored office-preparation bottle with a ground cap (Fig. 100). A small glass tube kept in the bottle readily assists in obtaining the few drops necessary for each treatment, to be placed upon an aseptic glass tray. Under no condition should pliers charged with cotton, etc., be introduced into the preparation in the bottle, and no unused portions of the solution must be returned to the stock-bottle.

The terminology of the substances which are used for the purpose of combating infection is frequently employed in a very loose manner, and without regard to its precise meaning. Hence, therefore, it may not be amiss to give as a preamble the definite significance of the more important terms as they are used to designate the specific nature of their action. It is generally recognized at present that the breaking down of highly organized bodies

is brought about by the activity of minute vegetable organisms—the bacteria. This process is known as putrefaction, or, under certain conditions, as fermentation. The presence of certain bacteria and their products is instrumental in the production of severe physiologic changes resulting in the various vital phenomena known as infectious diseases. The existence of a condition in which bacterial infection and its sequels are brought about by the presence of germs or their products is referred to as sepsis, while asepsis implies the entire freedom from such infection. If a primarily septic condition is changed by some method or means which inhibits the growth of the putrefactive organism, antisepsis is

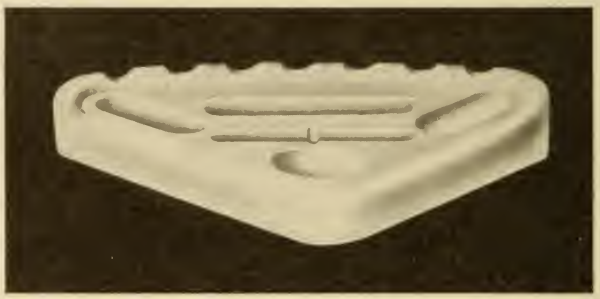
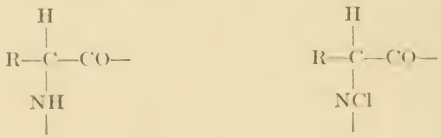


FIG. 101.—Aseptic medicament tray.

induced. Antiseptics, therefore, are chemical agents which merely inhibit the action and growth of bacteria, while germicides destroy the vitality of the infective organisms. Disinfectants also kill the bacteria, and incidentally chemically change their poisonous products to some inert compound. A disinfectant must, therefore, be a germicide, while an antiseptic is not necessarily a germicide nor a disinfectant.

According to Dakin and Dunham, the action of chlorin upon bacteria and their products seems to depend upon a process of chlorination, *i. e.*, the amino-acid group of the proteins readily



attack all substances containing "active" chlorin in such a way that the hydrogen attached to the nitrogen atom is replaced by chlorin. The newly formed compounds contain the NCl group, and therefore belong to the class of chloramins.

The chlorin of these newly linked compounds is still available, and these substances themselves are active germicides. A part of

the liberated chlorin is used by forming inert compounds, that is, chlorin unites with carbon to form inert chlorids. Incidentally, the liberated chlorin is a strong oxidizing (bleaching) agent which is a most beneficial factor in the treatment of pulpless teeth. Chlorin further acts as a prompt deodorizing agent, and possesses the additional remarkable properties of digesting and removing sloughing necrotic tissue and of decomposing toxins.

Dichloramin-T shares with other chlorin compounds the property of being a very active lymphagogue, *i. e.*, the amount of wound secretion, especially in the beginning of the treatment, may be considerably increased. The writer's attention has been frequently drawn to this fact by fellow practitioners who have tried the compound in treating root canals, and who complained of the increased secretions from the canals, which, incidentally, influences the granulation of the wound most beneficially.

The application of the antiseptic principle as utilized in wound sterilization depends primarily upon three definite conditions.

1. Absolute contact of the antiseptic with the infected organism.
2. Time during which this contact is maintained.
3. Sufficient concentration of the antiseptic at the point of contact.

Absolute contact between the antiseptic agent and the substances to be acted upon must be rigidly observed, as no antiseptic is known to act at a distance.

Finally, the permissible concentration of an antiseptic depends largely upon the tolerance of the tissues with which it is brought in contact, and is usually obtained from clinical observation. The concentration of the antiseptic solution determines its mass action which can be safely employed for tissue sterilization.

Concerning the relative bactericidal properties of the dichloramin-T solution, Drs. Lee and Furness make the following comment:

Theoretically, then, this new chlorin compound eliminated at the start the chief indication or necessity for the Carrel technic, skin irritation. With such a solution it should be possible to present to an infection an overwhelming mass of germicide, a 20 per cent solution of dichloramin-T being approximately eighty times the germicidal mass of 0.48 per cent hypochlorite solution. There is a vital necessity, when using germicides in the treatment of infections, for the earliest possible application of an overwhelming mass of a rapidly acting agent, because infection develops in the tissues at the rate of a geometric progression, and not by the slow process of addition, and therefore, every minute counts in the end result. Dichloramin-T, with a phenol-oil coefficient of about 50, can be presented in a larger mass without injury to the tissue cells than any other germicide we have used. Instead of having this chlorin given up with explosive rapidity and the consequent necessity of

frequent renewals of the solution, it would be slowly diffused into the surrounding media, making it unnecessary to renew the solution or to dress the wounds more frequently than once in every twenty-four hours. Consequently all dead tissue (in our particular instance, the débris of the gangrenous pulp) must be mechanically removed. Regarding the factor of time, it must be understood that the action of the antiseptic is enormously influenced by the medium in which it is dispersed, *i. e.*, a specific solution of one antiseptic may kill a given number of bacteria suspended in water very rapidly, while this same solution upon bacteria suspended in blood serum may be practically insignificant. The stability of an antiseptic as an inherent property is also of vital importance, since the innumerable changes which it undergoes during its action, absorption, etc., are of extremely wide variation. Dr. Dunham has very ingeniously depicted the speed of disinfection of a few widely used antiseptics in the following chart:

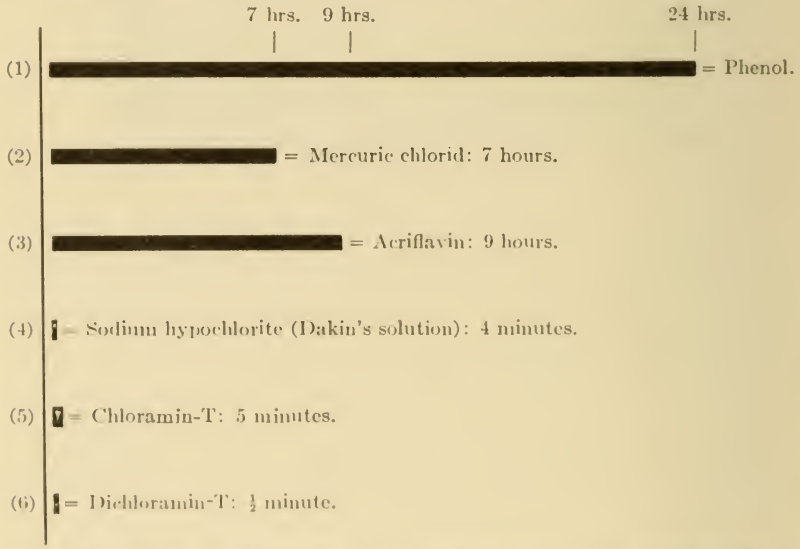


FIG. 102.—Speed of disinfection. A 2 per cent solution of (1) phenol failed to sterilize the mixture in twenty-four hours, although the surviving organisms were only 2 per 1000 of those originally present. (2) Mercuric chlorid, 1:1000, accomplished the same degree of disinfection in three hours, and completely sterilized in seven hours. (3) Acriflavin, a dye recently introduced as an antiseptic, 3:1000, killed all the bacteria in about nine hours. (4) Sodium hypochlorite (Dakin's solution), 0.5 per cent, completely sterilized in four minutes. (5) Chloramin-T in five minutes. (6) Dichloramin-T, 2 per cent in oil solution, in less than half a minute when well mixed with the septic material. (Dunham.)

Technic of Applying Dichloramin-T Solution to a Root Canal.—After the root canal has been suitably prepared by mechanical and chemical means so as to present a conical shaped tube, a freshly

flamed wire is inserted to the very apex and bent so as to form a shoulder near the pulpal wall. A roentgen picture is now taken. The tooth is again placed under rubber dam, the wire is removed and the canal is washed with sterile water and dryed out. Sterile paper points assisted by a few drops of aceton, absolute alcohol, etc., are serviceable for this purpose. Overheating of the tooth must be carefully avoided; however, a fair dryness of the root canal must be insisted upon. A suitable paper point is now saturated with dichloramin-T, carried to the root canal, and with a gentle pumping motion an attempt is made to coat the walls of the latter, and if possible a droplet is forced into the periapical space. The use of the warm air blast is of material assistance in getting the oily solution into the finer ramifications of the canal. The warm air blast is recommended in this connection solely for its mechanical effect in aiding the diffusion of the dichloramin-T over the dentin surface, but its use as a means for previous desiccation of the canal walls is not advisable, for the reason that the natural moisture of the tooth structure is necessary to the production of nascent chlorin resulting from the reaction of the dichloramin-T with the moisture of the organic structure of the tooth. A fresh point carrying a drop of the chlorin solution is now slowly forced into the canal to its very end and immediately sealed with a suitable retainer. As we have stated above, close contact of the antiseptic solution with the walls of the root canal, and if possible, with the surface of the involved infected area within the periapical tissues, is essential to obtain therapeutic results. The first application remains undisturbed for twenty-four hours. At the return of the patient the point is removed aseptically and carefully examined.

Fig. 103. — Absorbent paper point.

A second, a third, or, on rare occasions, a fourth dichloramin-T treatment is placed in the dry canal and these applications again remain respectively undisturbed for twenty-four hours. The paper cone which is removed at the last sitting must show no discoloration, it must have a distinct odor of chlorin and *not* of hypochlorous acid, and it must be fairly free from absorbed exudates. If possible, the treatments should not be left in the canal over twenty-four hours, at the end of this time the chlorin compound is completely exhausted, and usually a flow of lymph, as referred to above, is the sequence. Should the flow of lymph be rather copious, a dry, sterile paper cone may be inserted for a day or two under a hermetic seal; usually normal conditions of the periapical tissues will speedily return. If at the last treatment the canal is found satisfactorily

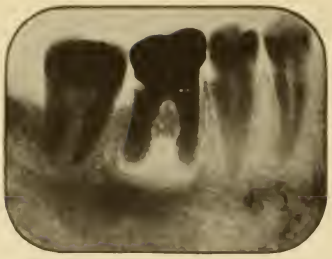


FIG. 104.—Lower left first molar carrying a gold shell crown. Tender on pressure; occasionally swelling near the apical region with paroxysmal pain at intervals. Large rarefied area near the apices of the two roots.

FIG. 105.—Same tooth after treatment with Dichloramin-T; four applications at intervals of about forty-eight hours.

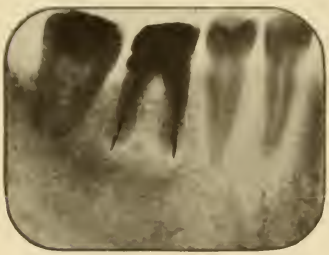


FIG. 106.—Bacteriologic examination of root canals negative; root canals filled.

FIG. 107.—Appearance of tooth and rarefied area one year after treatment.

FIG. 108.—First and second left lower premolars; sore on pressure and occasionally swelling.

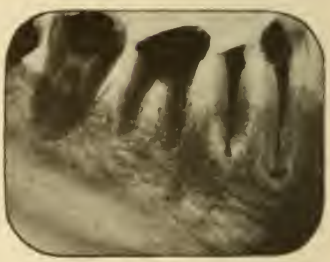


FIG. 109.—Appearance of teeth one week after treatment with three applications of dichloramine-T. Root canals filled.

FIG. 110.—Appearance of teeth one year after treatment. Roentgenogram shows eradication of infected apical foci.

clean a microscopic examination in the form of a smear obtained from the removed cone is made. If the examination indicates sterility, no time should be lost in filling the canal at once.

Regarding the existing sterility of a primarily infected root canal as treated by the above-outlined dichloramin-T method, it should be emphasized that rigorous bacteriologic tests were made in numerous instances in the routine way by plating out scrapings from incubation upon agar plates in bouillon, etc. After exposure in an incubator for various lengths of time, usually from forty-eight to seventy-two hours, it was observed that bacterial growth from the previously infected canals was negative, *i. e.*, no cultures were obtained usually after the third, and, in a few cases, after the fourth treatment.

ELECTRO-STERILIZATION OF INFECTED ROOT CANALS.

History.—The utilization of the electric current for the purpose of checking bacterial growth is by no means of recent origin. In 1883 Cohn and Mendelsohn employed a galvanic current experimentally to study its effect upon bacteria suspended in nutrient solution. Apostoli and Laquerrière, in 1890, used a current of 100 to 150 milliampères for similar purposes, claiming, however, that it is not the current as such but the electrolytic decomposition of the nutrient fluid which produces the desired effects. Similar results were obtained by Prochowink and Spaeth in the same year. In 1891 Versoogen claimed that the bactericidal effect of the current depended on the end products of the electrolytic dissociation of a suitable fluid, *i. e.*, acid at the positive pole and alkali at the negative pole. The application of this principle for the purpose of sterilizing root canals was probably first attempted by Breuer, of Vienna, who, as early as 1890, referred to this method in a tentative manner. In 1895 Rhein, at the suggestion of Morton, employed this procedure in an empiric way with apparent good success; he again demonstrated it in 1897, and has referred to it at various times ever since. A systematic investigation of this procedure was first attempted by Bethel in 1896–1897, and a careful perusal of his publications is of the highest interest. However, it remained for Zierler to furnish a detailed account of the nature of the action of the galvanic current on bacterial infection of root canals, and his work, carried out in conjunction with Lehmann in 1900, must be regarded as a very complete *exposé* of the procedure. Since then quite a host of writers have elaborated on this problem, among whom Hoffendahl, Miller, Peter, J. Forbes Webster, Frank D. Price, Sturridge and many others should be mentioned. The dental profession is particularly to be congratulated on the

excellent discussion of this procedure as recorded by Sturridge in his commendable work *Dental Electro-therapeutics*.

The term ionization as applied to the specific purpose of electro-sterilization of root canals is ill chosen. Ionization designates purely a chemico-physical process, *i. e.*, by the phenomenon of electrolysis ions are induced to migrate. Again, the term ionic medication is employed for such procedures. Lewis Jones defines it as follows: "Ionic medication is a method of treatment in which electric currents are used for their power of setting the constituents of a saline solution in orderly motion in a definite direction. It is used for the introduction of drugs into the superficial parts of the body through the surface." While the above explanation is compatible with the nature of ionic medication for general medicinal purpose, it does not specifically designate the intent for which this procedure is applied in the treatment of root canals. The sole object of this treatment consists in combating infection by the most powerful method known, *i. e.*, sterilization. The latter is induced by a weak electric current through the migration of certain ions. Hence the term sterilization by electrolysis, or in short, electro-sterilization, as first suggested by Zierler, is eminently suitable for this purpose, and its general adaptation is recommended by the writer. It has been suggested that the term electro-sterilization merely indicates a substitute for the once famous therapeutic procedure known as cataphoresis. This conception is not correct. Cataphoresis designates the mechanical movement of suspended molecules by means of the electric current. As such it is a process independent of electrolysis. The rationale of electro-sterilization depends on the interaction of two definite processes: (1) The dissociation of a suitable chemical compound in a solvent (electrolyte) into ions, and (2) the movement of these ions in the direction of specific poles within the tissues, brought about by the passage of a weak galvanic current.

Theory of Electrolytic Dissociation.—When a solid, liquid or gas enters into solution and is capable of conducting an electric current, according to Arrhenius, the solution undergoes certain changes which are grouped under the generic term electrolysis. This latter term and the following nomenclature was introduced by the English physicist Faraday (1791–1867) and is still universally employed. The solution itself is known as the electrolyte, while the dissociated products are referred to as ions. The terminals at which the electric current enters or leaves the electrolyte are called electrodes. An ion (ion = going) may be referred to as being the dissociated product of a chemical decomposition which is capable of conducting an electric charge, and which travels in the direction of an oppositely charged pole. Those ions which are charged negatively migrate to the anode, *i. e.*, the positive pole, and are known as anions, while

the positively charged ions migrate to the negative pole, the cathode, and are known as cations. Relatively speaking, all metals, alkaloids and hydrogen, are positive ions, *i. e.*, cations, while all acids, bases, halogens, hydroxyl compounds and oxygen are negative ions, *i. e.*, anions.

As Ostwald has suggested, the cation may be designated by the positive sign $^{+}$ or by ˙, and the anion by the negative sign $^{-}$ or by ′.

TABLE OF IONS, THEIR ELECTRO-CHEMICAL EQUIVALENTS AND RELATIVE VELOCITIES ACCORDING TO LEDUC.

Ions.	Milligrams per coulomb.	Milligrams per milliampère-minute.	Relative velocities.
Anions:			
Bromin	0.82	0.049	0.9
Chlorin	0.367	0.022	1.0
Hydroxyl	0.18	0.01	1.27
Iodin	1.31	0.078	1.16
Salicylic acid	1.4	0.085	
Cations:			
Ammonium	0.06	0.003	1.56
Calcium	0.206	0.012	0.5
Cocain	3.0	0.18	0.59
Gold	0.678	0.04	1.22
Hydrogen	0.01	0.0006	0.88
Lithium	0.07	0.004	1.28
Magnesium	0.115	0.007	0.5
Mercury	1.03	0.062	0.8
Potassium	0.4	0.024	1.0
Quinin	3.9	0.234	0.62
Radium	1.13	0.066	
Silver	1.1	0.06	0.5
Sodium	0.23	0.014	1.6
Strychnin	3.4	0.207	
Sulphur.	0.16	0.01	
Zinc	0.33	0.02	0.6

"An ion may be either a charged atom, as in the case of the silver ion, or a charged group of atoms, or molecules. In the case of silver nitrate, $AgNO_3$, the cation is Ag, and the anion is the molecule or radicle NO_3. The charge of the NO_3 ion is one negative unit, and that of the Ag ion is one positive unit, as both the ions are monads, or monatomic." (Lewis Jones.)

A simple solution of salt in water dissociates the salt into electro-molecules, the ions, which exist independently of the action of a galvanic current. The number of positively and negatively charged ions is equi-molecular, *i. e.*, the solution is electrically neutral. The ions themselves are suspended in the solution in a chaotic mixture. The passing of the galvanic current, according to Nernst, by its electro-motive force causes a definite movement of the ions in an orderly direction to their specific centers of attraction; *i. e.*, respectively to the positive and the negative pole.

The nature of the movement of ions may be theoretically explained according to Nernst, by the schematic drawing, Fig. 111.

According to this scheme, if two zinc electrodes are charged, *A* and *B*, suspended in a zinc chlorid solution, *D*, from battery *C*, the positive zinc ions, *F*, are attracted to the negatively charged electrode, *B*, and slowly they move in the direction of this pole. The zinc ions discharge their positive charge, thereby releasing the negatively charged electrode. The discharged zinc ions are now formed into ordinary metallic zinc. The positively charged electrode, *A*, attracts the negatively charged chlorin ions, *E*; the latter discharge themselves and dissolve metallic zinc, forming zinc chlorid, which at once is again ionized. The resultant ions—zinc and

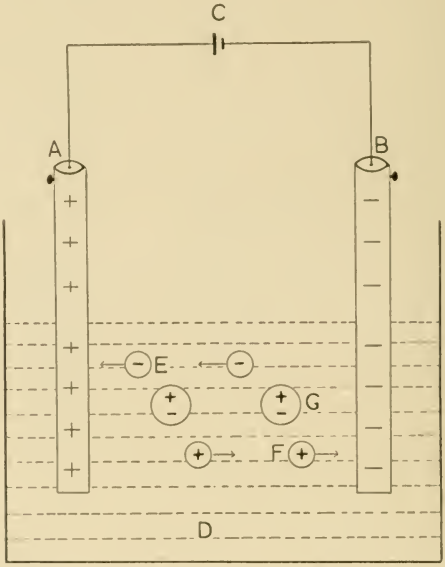


FIG. 111.—Scheme showing the movement of ions.

chlorin—are dispersed in the solution. Undissociated molecules, *G*, of zinc chlorid present in the solution are not acted upon by the current. The movement of these ions occurs comparatively slowly and may be accurately measured by means of an ingenious apparatus devised by Kohlrausch.

The degree of concentration of the solution to be ionized has no effect upon the number of ions produced; the latter depends upon the strength of the current multiplied by the time for which it is applied. In other words, ionization is a manifestation of transformed electric energy in accordance with Faraday's law. The amount of decomposition of an electrolyte is proportional to the amount of electricity which flows through it.

The process of electro-sterilization of infected root canals concerns itself primarily with the disinfectant action of the liberated ions, and less so with their supposed medicinal qualities. The disinfectant action is principally confined to the surface of the object treated although a certain depth of penetration is desirable. According to Sir Oliver Lodge, chlorin the principal agent depended upon in root-canal sterilization develops a velocity of penetration amounting to 2.16 mm. per hour for a drop of potential of 1 volt per cm.

The electric current *per se*, *i. e.*, at least in the strength suitable for root-canal sterilization, does not produce any measurable bactericidal action. A weak current passed for hours through diluted sulphuric acid prior to entering an inoculated Petri dish does not inhibit the growth. In the presence of an electrolyte, the current acts on the dissociated ions of the latter, and depending upon their specific chemical nature, some of the most powerful disinfectants may be obtained. It is claimed that certain pure metals as such possess slight antiseptic action. This property was first observed by the late Professor Miller. According to Behring, this antiseptic action is the result of the reaction of certain waste products of bacteria, primarily lactic acid, with those metals which are capable of forming soluble salts, and which diffuse through the medium. This antiseptic action of metals must not be confounded with the oligodynamic action of certain pure metals in their colloidal state, as copper, for instance, on low-type plant cells. Of the pure metals, according to the classic experiments made by Thiele and Wolf, mercury, silver and copper are the only ones which produce poisonous salts in the presence of bacteria, while the other tested metals, *i. e.*, platinum, palladium, gold, aluminum, magnesium, zinc, lead, tin and iron are wholly devoid of action. In the discussion of electro-sterilization of infected root canals, great stress is frequently laid upon the specific nature of the metallic electrode placed in the root canal as being *the* factor which produces the desired germicidal effect. Rhein, for instance, insists on using a chemically pure zinc electrode in the presence of a sodium chlorid solution, claiming that "nascent zinc chlorid" is formed during the process of electrolysis. Other practitioners employ a copper electrode and a weak zinc chlorid solution as a substitute for the sodium chlorid solution. A zinc electrode employed for electro-sterilization of root canals is not only devoid of any germicidal action, but it is also an ill-chosen metal for this purpose, because a zinc wire is too brittle to be filed fine enough so as to readily enter a minute root canal without inviting danger of breaking.

Ionization of a metallic electrode occurs primarily in the presence of a suitable electrolyte, *i. e.*, a solution of a salt of the metal of the respective electrode. While theoretically it is true that ions

of the respective electrode must be produced as a secondary sequence of the primary ionization of the electrolyte, *practically*, in employing the low amperage tolerated by the human body these ions are *not* demonstrable with the ordinary chemical reactions, consequently, they cannot exercise any therapeutic effect. A zinc electrode in the presence of a sodium chlorid solution is *not* ionized in the short space of time and with the low amperage employed in the electro-sterilization of root canals, consequently "nascent zinc chlorid" ions, which are believed to have been produced from zinc electrodes, are imaginary therapeutic bodies. When a high amperage is employed—in experimental work outside of the human body—sufficient hydrochloric acid is obtained as a secondary product which will act on the zinc pole, forming zinc chlorid.

The Electric Current and Its Accessories.—The only current suitable for electro-sterilization is the direct current. The alternating current as such cannot be used unless it is changed by a transformer. This may be accomplished by a chemical "rectifier" or a small motor dynamo. The chemical rectifier without potential equalizer has not been found satisfactory by the writer. The source of the current may be obtained from the main line, from an accumulator or a storage battery, or from a series of cells. If the street current is used, it must be reduced by a rheostat to about 30 to 40 volts. A number of lamps, mounted in series, one lamp of sufficiently high voltage, or a wire rheostat, is usually employed for this purpose. An ordinary switchboard is less suitable, as there is always danger of shocking the patient through imperfect control. If the street current is used a knife switch should be interposed between the rheostat and the current controller. If cells are employed—and many practitioners and most of the reliable electric supply houses regard a cell series as the safest means for the purpose in view—about 18 to 24 Leclanché wet cells or an equal number of ordinary dry cells (Columbia No. 6) are most useful. The silver chlorid cell is less serviceable for our purpose. An ordinary wet or dry cell furnishes approximately a little over 1½ volts. Recently, compact types of dry-cell batteries furnishing a current of very low amperage and medium voltage, intended for wireless telegraphy, have been placed on the market. These cells are also useful for dental electro-sterilization. The cells are mounted in series and connected to binding-posts. From these posts the current is conveyed by means of flexible conducting cords to a suitable controller. The most important feature of a serviceable controller consists in the gradual increase or decrease of the current in very small fractions of a milliampère without shocking the patient. A graphite or a series wire rheostat, either plain or as a shunt, is serviceable for such purposes. The markings on the current controller (Fig. 112), be they volts or arbitrary numbers, have little bearing on the practical application of the current.

The current controller, in turn, is connected with a milliampèremeter, an instrument for measuring the quantity or strength of the current. The milliampèremeter is *the* instrument of precision

FIG. 112.—The S. S. White current controller.

which guides the operator in his work, consequently too much emphasis cannot be placed upon the importance on obtaining a perfect working instrument.

FIG. 113.—Weston milliampèremeter.

At this point the writer may be permitted to digress for a moment from the subject proper and call to the mind of the reader the fundamental nomenclature governing electrical measurements—as far as it is utilized in the following discussion. By the term ampère is

meant the unit of strength of a current. A milliampère is a $\frac{1}{1000}$ part of an ampère, expressed as M.A. A volt is the measure of the unit of pressure of the current, *i. e.*, the electric power necessary to drive a current of 1 ampère through a resistance of 1 ohm. It is referred to as the electro-motive force and expressed as E.M.F. An ohm measures the resistance of a circuit through which a current flows and a watt represents the units of power. The current strength flowing in a circuit is equal to the pressure divided by the

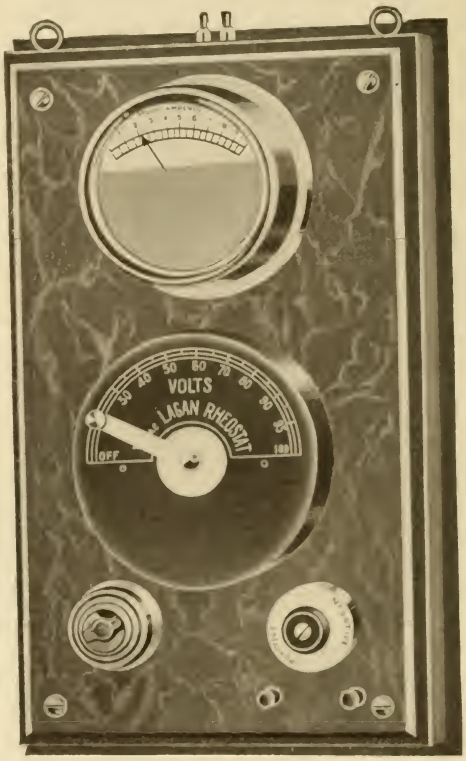


FIG. 114.—Switchboard for electro-sterilization. (McIntosh.)

resistance. The resistance equals the pressure divided by the strength. The pressure equals the strength multiplied by the resistance. Therefore, in simple terms:

Ampère	=	volts	÷	ohms.
Ohms	=	volts	÷	ampères.
Volts	=	ampères	×	ohms.
Watts	=	volts	×	ampères.

From the above explanation as related to the process of electro-sterilization, it is obvious that the correct measurement of the

amount of current applied to a patient is of the utmost importance, as it is the safest means of guiding us during its application. Hence the importance of procuring a trustworthy milliampèremeter. The best instruments are those constructed after the Deprez-d'Arsonval deadbeat (non-trembling) type. The Weston milliampèremeter (Fig. 113) is a most reliable current gauge. The face of the latter instruments, suitable for this work, should be calibrated in 5 milliampères, with subdivisions of $\frac{1}{10}$ to $\frac{1}{20}$ milliampère. To convey the current to the patient, different colored flexible cords are employed which terminate in suitable electrodes. In connecting up the whole apparatus extreme care must be observed in joining equal poles to each other; viz., positive pole must be connected to positive pole, and *vice versa*. To locate the

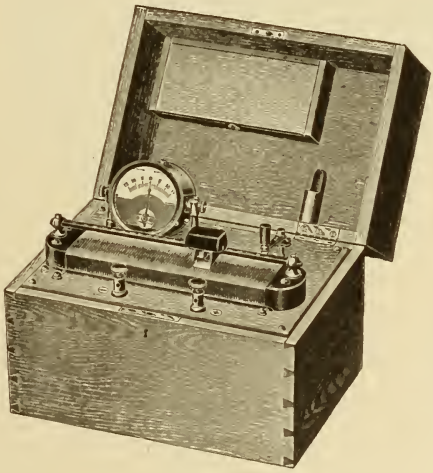


FIG. 115.—Galvanic battery for electro-sterilization.

respective poles, the following simple experiment may be employed. Moisten a piece of blue litmus paper with water. Place the two poles of the battery about 1 inch apart on the wet paper and turn on the current. In a few moments a pink spot will develop where the *positive* pole touches the paper.

The two electrodes are terminals attached for the purpose of conveying the current to the patient, and consist of a negative electrode which is to be placed on the patient's skin surface, and a positive electrode to be introduced into the tooth. The negative electrode may be a piece of metallic tubing held firmly in the patient's hand, or a sponge electrode fastened to his wrist, or one of various modifications thereof. The size of the negative hand electrode is important; it should present at least five square inches

surface area, which are to be brought into contact with the patient. A large surface of the negative electrode reduces the resistance, and consequently the tingling sensation or even blistering caused by the heat of a small electrode is avoided. The writer prefers the

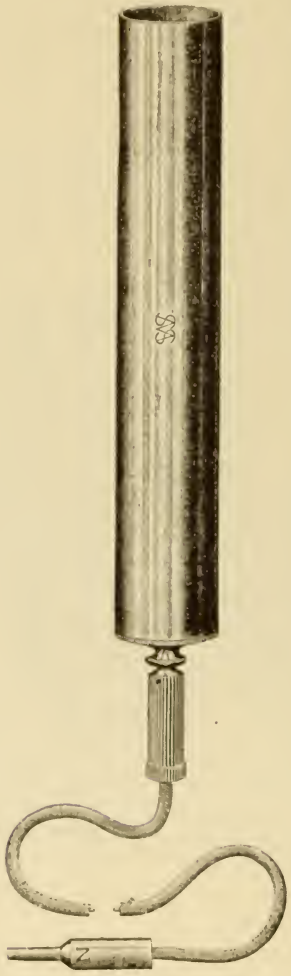


Fig. 116.—Metal negative hand electrode.

plain tube hand electrode, as it avoids the cumbersome wetting with salt water, loss of time in adjusting it, etc. It is immaterial in which hand the electrode is held. Rings, bracelets, wrist-watches, etc., must be removed, otherwise blistering of the patient's skin by mere contact may occur. To place the negative electrode

upon the patient's cheek, lip or gum surface by means of a clamp or spring, as recommended by some operators, is to be avoided, for the reason that severe burns may result. It has been stated that this blistering results from the formation of caustic sodium hydroxid

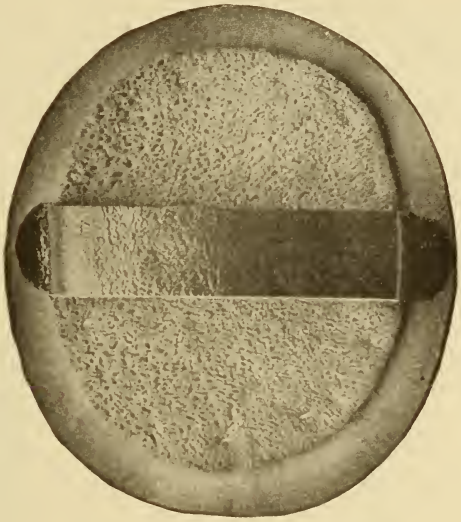


FIG. 117.—Sponge hand electrode.

near the negative pole. The blistering is the result of imperfect contact between the skin and the metal electrode, thereby increasing the resistance of a small area to such an extent as to produce high heat, *i. e.*, an electric burn. The positive electrode to be introduced into the tooth consists of a piece of iridio-platinum wire No. 20 gauge, about one inch long and tapered to a delicate point.

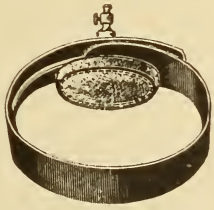


FIG. 118.—Sponge wrist electrode.

The iridio-platinum alloy possesses the necessary flexibility, which is lacking in pure platinum. The point itself is ground blunt so as to avoid being caught when introduced into tortuous canals. Various sizes of these points may be kept on hand. No other

metal should be employed for such purposes. To substitute the iridio-platinum point by zinc, copper, or any other metal with the view of aiding its therapeutic effects is not only useless but it markedly interferes with the action of electrolysis in the relatively small area of a root canal, or the resultant ions may discolor the tooth. A long-handle electrode holder, insulated with hard rubber is essential to suitably unite the electrode with the conducting cord. The holders may be of various types so as to give ready access to all parts of the oral cavity. From the foregoing descrip-

Fig. 119.—Long-handle electrode with iridio-platinum point.

Fig. 120.—Insulated electrode holder.

tion of the source of the current, its control and its mode of application, it may be observed that essentially it is a duplicate of the armamentarium as applied in producing cataphoresis. Any apparatus, therefore, that is or has been used for inducing cataphoresis may be equally successfully employed for the electro-sterilization of root canals.

Electro-sterilization Equation.—In the various communications treating on root sterilization by electrolysis the very important questions concerning the *time* during which the current is applied, the *number of milliampères* employed and *bacteriologic tests* of the

resultant sterility are usually vaguely treated. When sterility of a primarily infected root canal is spoken of in the present light of bacteriologic knowledge, the truth of this assertion has to be proved by rigorous tests, otherwise the term sterility loses its significance. These tests are readily made by obtaining cultures at stated intervals from the canal under treatment until complete negative results of growth are obtained. Regarding the bacteriologic tests as applied to electro-sterilization, the author proceeded as follows: Cultures of the infected root canal were made before treatment was instituted, and then every five minutes thereafter for a given period of time, usually twenty minutes. The infected agar plates were incubated in the routine manner (see below). Incidentally, the time of applying the current, and also its strength, were carefully noted. By comparing the results obtained, a definite relationship between the strength of the current, the time of application and the resultant sterility could be established. Zierler deserves credit for having first noted the inter-relationship of these factors, and he had suggested the use of a numerical constant which furnishes a working basis for its clinical application. This constant is 30. By multiplying the number of milliampères employed by the time in minutes used in the process of obtaining a sterile root canal, invariable a number was obtained which closely hovered about the figure 30; or, reversely, by dividing the constant 30 by the number of milliampères employed, a quotient is obtained which gives the time in minutes during which the current must be applied. Apparently, a given infected surface area requires for its sterilization, a specific amount of migrating ions; at least this assertion can be verified as far as the germicidal action of ionized chlorin is concerned in the sterilization of infected root canals. Hence the numerical constant 30 may be looked upon as expressing in units the surface area of an average root canal. In the author's experimental work and in clinical practice he has based his observations upon the above principle, and has collected sufficient data as proofs that the appended electro-sterilization equation, as this formula has been termed, is a reliable guide for the application of these procedures in the treatment of infected root canals: $\frac{30}{\text{M.A.}} = \text{T}$, the 30 representing the numerical constant, M.A. the number of milliampères, and T the time in minutes.

The tables on page 212, selected from experimental records, will substantiate these claims.

If the three constants which show sterility = $102\frac{1}{2}$ are added, and the total divided by the number of patients, the average constant will be 34, which in round figures may be reduced to 30. Various attempts were made to materially lower this constant, but so far (as regards sodium chlorid solution) have not been successful.

TABLE A.—ELECTRO-STERILIZATION OF TEETH IN THE MOUTHS OF PATIENTS.

Patient.	Milliampères.	Electrolyte.	5 min.	10 min.	15 min.	Constant.
No. 1	0.5	1 per cent sod. chl. solution	+	+	+	0
No. 2	2.5	"	+	+	0	372
No. 3	3.0	"	+	0	0	30
No. 4	3.5	"	+	0	0	35
No. 5	1.5	"	+	+	+	0

Growth +. No growth 0. The inoculated agar plates are incubated at twenty-four hours at 40° C.

TABLE B.—ELECTRO-STERILIZATION OF EXTRACTED TEETH HAVING GANGRENOUS ROOT CANALS.

Teeth.	Milliampères.	Electrolyte.	5 min.	10 min.	15 min.	25 min.	Constant.
No. 1	0.5	1 per cent sod. chl. solution	+	+	+	+	0
No. 2	1.0	"	+	+	+	0	25
No. 3	2.0	"	+	+	0	0	30
No. 4	3.0	"	+	0	0	0	30
No. 5	5.0	"	0	0	0	0	25

Growth +. No growth 0. The inoculated agar plates are incubated for twenty-four hours at 40° C. Total constants: 110 : 4 = $27\frac{1}{2}$ average constant.

TABLE C.—ELECTRO-STERILIZATION OF PLATED MIXED CULTURES OBTAINED FROM GANGRENOUS ROOT-CANALS IN AGAR.

Plate.	Milliampères.	Electrolyte.	5 min.	10 min.	15 min.	Constant.
No. 1	1.0	1 per cent sod. chl. solution	+	+	+	0
No. 2	2.0	"	+	+	0	30
No. 3	3.0	"	+	0	0	30
No. 4	4.0	"	+	0	0	40

Growth +. No growth 0. The inoculated agar plates contain 1 per cent sodium chlorid and are incubated for twenty-four hours at 40° C. Total constants: 100 : 3 = $33\frac{1}{3}$ average constant.

The above experimental work furnishes sufficient proof, and this fact is borne out in clinical practice, that sterility of the walls of an infected root canal may be obtained by electro-sterilization.

It should be borne in mind, however, that this particular type of sterilization means *sterility of the walls of the root canals only* and *not* of the entire tooth root. To prove the correctness of this statement, which on its very face is self-evident, tooth roots Nos. 2, 3 and 4 of the experiments recorded in Table C were cut into coarse shavings, plated on agar, and placed in the incubator. In twenty-four hours all three plates showed luxuriant growth. Here will be emphasized again what has been stated in the past at frequent intervals: Complete sterilization of an infected root *in situ* is impossible by any of the present known methods; complete sterilization of the surface of an infected root canal is possible by electro-sterilization as outlined above.

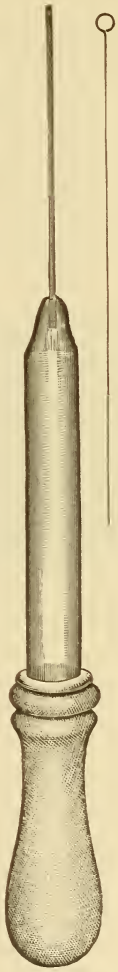


Fig. 121.—Minim syringe for applying saline solution.

Clinical Application of Electro-sterilization.—To convey to the reader a practical working knowledge of the clinical application of the principles of electro-sterilization, it is probably best to describe the actual *modus operandi* in detail as employed in a typical case. The patient being seated in a chair is covered by a rubber apron sufficiently large to reach over the chair arms, so as to protect him from accidental shock by "grounding" the current. The root canal of the tooth to be treated must be mechanically cleansed of its débris, and if necessary enlarged so as to give free access to the wire electrode. Before starting the ionizing process it is best to assure oneself of the correct working of the current by bringing the two poles together for a moment; the moving of the needle of the milliampèremeter in the right direction acts as an indicator that the apparatus is in working order. The rubber dam having been adjusted, the root canal is now flooded with a 1 per cent saline solution—an S.S.W. minim syringe is useful for such purpose. The patient takes a firm hold of the negative electrode with his hand, which must not carry rings, bracelets, etc. Before introducing the freshly flamed positive pole into the canal the operator should see to it that the knife switch is open, and that the controller is set at zero. If the wire electrode fits the canal too loosely, a few fibers of cotton moistened with salt water are wrapped about it. The needle is introduced as near to the apex as possible, and the knife switch is closed. The controller is now very slowly

turned on, and the patient is told to at once raise his hand when he feels the slightest sensation. The moving needle of the milliampèremeter will indicate to the operator that the current is flowing in the right direction. When the patient raises his hand the controller is turned very slightly back, left at this point for about half a minute, and again very slowly turned forward until the patient again responds, or until the point of tolerance is established. This point the writer has termed the "irritation point." A glance at the milliampèremeter conveys to the operator the number of milliampères employed. The operator now recalls to his mind the numerical constant 30, and quickly calculates the time of his particular case of electro-sterilization by dividing 30 by the number of milliampères employed. The resultant quotient gives the time in minutes for which the current must be applied. Example: If the patient's irritation point is 2.5 M.A., twelve minutes by the

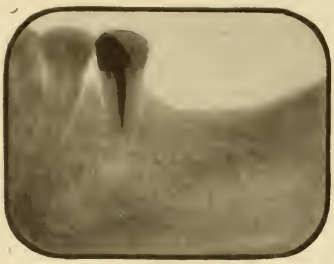


Fig. 122.—Lower right second premolar. Root canal is imperfectly filled. A rarefied area is visible about the apical end. Continuous gnawing pain on pressure.

Fig. 123.—Same tooth as in Fig. 122. Root canal treated by electro-sterilization; root canal filled. Former trouble has completely subsided. Roentgenogram taken a year later; tooth in perfect condition.

watch are required for the sterilization of this particular root canal. If the resultant quotient is a fraction the writer recommends that the next higher unit be substituted as the indicator of the time. Each root canal of a multi-rooted tooth is preferable treated separately. If a clamp electrode holder is employed to clasp the two or three wires inserted into the multi-rooted tooth care should be exercised to prevent short-circuiting. To avoid polarization of the positive electrode, *i. e.*, covering by a film of nascent gases which materially interferes with the flow of the current, the needle should be removed at five-minute intervals (turn off current previously!) and wiped off. During the process of electro-sterilization a drop of salt water should be added about every minute to make up for loss of evaporation. Care must be exercised to prevent short-circuiting of the current by allowing salt water to seep under the rubber dam and thus transfer the cur-

rent to the gum tissue. After finishing the operation the controller is slowly turned to zero, the knife switch is opened, and the electrode removed from the tooth. Never remove the electrode without having first cut off the current, otherwise the patient receives a disagreeable shock or a flash of light passing in the eyes. On passing a few fibers of cotton or a paper cone in the root canal, a pronounced odor of chlorin should be perceptible. A wisp of cotton or a cone wet with salt water is placed into the root canal, and the latter is closed with gutta-percha stopping. The treatment is to be repeated within twenty-four hours, and if necessary again on the third day, and the canal is immediately filled after the last treatment. A root canal should never be filled immediately after the initial treatment; an interval of at least twenty-four hours should be allowed before doing so. Migrating ions do not develop their maximum degree of therapeutic efficiency within the short

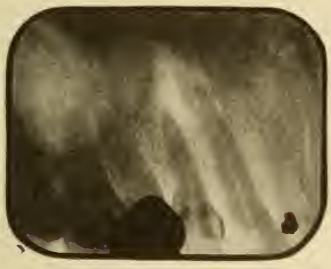


Fig. 124.—Upper left first premolar. Chronic pericementitis with obscured area about the foramen; root canal filled with pus. Continuous pain for over a week.

Fig. 125.—Same tooth as in Fig. 124. Root canal treated by electrosterilization; root canal filled. Roentgenogram seven months later; no further trouble observed.

period of time during which the current is applied. It requires practically twenty-four hours to produce their full activity within the region of a root canal and its surroundings. The clinical indications of complete sterility are definite odor of chlorin and a clean paper or cotton cone after twenty-four hours' insertion. In doubtful cases sterility should be verified by a bacteriologic test. If a metal filling is present in the tooth under treatment it should be removed, because if touched by the electrode after the current is turned on it may be short-circuited through the filling, and the patient will receive a shock. Moreover, the action of the chlorin ions upon the metals of the filling materials results in the formation of metallic chlorids, which infiltrate the dentin structure, producing discoloration. This is particularly true in the case of gold chlorid thus formed, which by secondary decomposition stains the tooth structure a deep purple tint.

When the products of pulp decomposition pass beyond the

foramen of a tooth, localized pathologic disturbances of the pericementum arise, which usually lead to the formation of an abscess. Without entering into the further discussion of the pathology of the disturbances at this moment, let us assume that the disturbances are eradicated by establishing drainage along the lines of least resistance. If the drainage takes place through the root canal this condition is spoken of, although wrongly, as a blind abscess, while if the drainage occurs through an artificially established canal through the bone and gum tissue a fistula results. Acute types of the enumerated disturbances yield readily to electrosterilization, provided the salt solution and the positive electrode reach the seat of the infection. For the treatment of an abscess draining through the root canal the positive electrode is thrust through the foramen into the abscess cavity; the treatment of an abscess with a fistula requires a somewhat modified application. In the latter case complete communication between the root canal and the mouth of the fistula must be first established by forcing warm salt water through the canal. The root canal is now treated as outlined above; the fistula itself requires a separate application of the procedure. The positive electrode is passed into the fistula, entering at its outlet and carried along the fistulous tract until the root is felt, while the negative pole, consisting of a piece of copper wire surrounded by salt water, is placed in the root canal. The sterilization equation for this treatment is the same as already outlined. Usually the patient requires a lower milliampèrage for such work. All types of chronic abscesses will yield to this method of treatment, provided the necrotic area involved is very small, and that the seat of disturbance is reached by the electrode and by the salt water.

SILVER NITRITE IN THE STERILIZATION OF INFECTED ROOT CANALS.

Silver nitrate as a means of sterilizing infected root canals has been recommended at various times during the last decades. A most excellent discussion appeared by Bethel,[1] in 1898, and ever since, sporatically, it has been advocated anew. The solutions employed for this purpose vary greatly in regard to their strength and relative to the nature of the reducing agents employed. The impregnation of the walls of a root canal or of carious defects with silver nitrate unquestionably permanently inhibits further microbal infection. The process may be explained as follows: The favorable action of argentic nitrate on the course of caries is obtained by the insoluble combinations which it forms with the organic

[1] Ohio Dental Journal, 1896, No. 9.

tooth substance, and thus withdrawing the nourishment from the bacteria. We may assume that the chemical process consists in the coagulation of the albumin by the liberated nitric acid and the formation of insoluble silver albuminate and of silver oxid. Furthermore, since the animal tissues always contain sodium chlorid, a further chemical change occurs, in which the nitric acid ion of the argentic nitrate combines with the sodium and the chlorin combines with the silver to form the insoluble silver chlorid:

$$AgNO_3 + NaCl = AgCl + NaNO_3$$

Miller has proved, however, that this newly formed insoluble silver chlorid does not resist the action of acids in any marked degree, but that it is the solid mass of precipitated black silver-albumin chlorid which acts principally as the resisting force. This explanation corresponds with clinical observations. If silver nitrate is placed into a cavity or upon tooth structure and is immediately covered by a protective layer of gutta-percha or cement its action is largely nullified; only a lemon-yellow stain—xanthoprotein—results. On exposure to light, the yellow color changes to a jet-black stain which now is immune to caries. Hence the significance of Black's dogmatic postulate: "Expose the tooth surface treated with silver nitrate to sunlight for ten minutes until a full black is obtained." The many offered substitutes for silver nitrate, *i. e.*, silver lactate and citrate, or the colloidal silver and the organic silver compounds are of little practical use for this purpose.

Shanasy, in 1910, advocated formaldehyd and caustic potash as reducing agents while Howe[1] employs an ammoniated silver nitrate solution reduced by formaldehyd, and Rickert[2] employs a moist silver oxid precipitated by the hydroxid of potassium or sodium. Again, other practitioners have used essential oils, such as oil of clove, eugenol, etc., for the same purpose. Howe employs the following procedure:

"Two solutions are required.

"*Solution* 1.—This consists of a saturated solution of silver nitrate in water to which is added little by little strong ammonia. As the ammonia is added a dark precipitate of silver oxid is thrown down. This is soluble in an excess of ammonia, therefore continue adding the ammonia until the solution becomes clear.

"*Solution* 2.—This consists of a 25 per cent solution of formalin in water.

"These two solutions must be kept in separate dark-colored bottles, with glass stoppers, and should be kept away from the light

[1] Dental Cosmos, 1917, p. 891.
[2] Journal of the National Dental Association, 1919, p. 930.

as much as possible. They work better if they are freshly prepared, but are still good after a considerable time, if kept as recommended.

"The principle employed is that of reducing the silver from its solution. Metallic silver is thrown down in a very finely divided state. It is deposited upon the sides of a clean test-tube as a mirror. The principle is used in photography and in staining methods in histology. The action in the tooth is the same. A finely divided deposit of silver in its metallic form occurs wherever

FIG. 126

FIG. 127

FIGS. 126 and 127.—Teeth treated with silver nitrate by the Howe method. The healthy tissue is not penetrated by the silver solution, but the infected structure is. (Howe.)

the liquid penetrates. By successive applications a very appreciable thickness of deposit occurs. This may be burnished and made to take on the luster of the metal. The reaction in this method is as follows:

$$2AgNO_3 + 2NH_4OH = Ag_2O + 2NH_4NO_3 + H_2O$$
$$Ag_2O + CH_2O + 2Ag + HCOOH$$

"Formic acid acts readily as a reducing agent, taking away oxygen and forming carbonic acid, H_2CO_3, which decomposes easily into carbon dioxid and water. We have then practically metallic silver and nothing else. The reaction sterilizes, as we have ascertained by repeated examination and at the same time leaves this heavy deposit of metallic silver in a fine state of subdivision, which penetrates all affected dentin but does not penetrate the sound tissue of the teeth.

"The method of procedure is very simple. Such slight changes as are needed to make it adaptable to different localities in the mouth can readily be solved by the ingenuity of the operator.

"It is better to apply the rubber dam or to protect the tissues in some way. Any applicator will answer the purpose for conveying the liquids to the cavity. Broaches wrapped with cotton will

serve the purpose or glass tubing drawn out to a capillary end may be used. These we prepare by heating a piece of small-bore glass

Fig. 128

Fig. 129

Fig. 130

Figs. 128, 129 and 130.—Illustrating the penetration of the silver solution to the very end of the roots, even when the broach is unable to reach it. (Howe.)

tubing in the middle and drawing it out to a capillary. With a file this is then broken in the center. One tube we keep for the ammoniacal silver solution and the other for the formalin. If the

Fig. 131

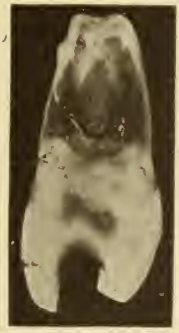


Fig. 132

Fig, 133

Figs. 131, 132 and 133.—Localized areas of dentin which have become hyaline are not stained by the silver nitrate solution; the tubules are absolutely sealed. (Howe.)

solutions are of sufficient depth in the stock bottles the liquid will collect in the capillary end of the tubes, or it can be drawn up into

the tubes by suction. The fluid can be retained and controlled by placing the finger over the large end of the tube. Tubes with curved ends are used for the upper teeth.

"A tube of Solution 1 is taken, the capillary portion filled, the finger placed over the end and this is conveyed to the cavity. By momentarily raising the finger a small drop of the silver solution is allowed to flow into the tooth. A small drop of Solution 2 is now flowed in, the solution darkens, silver is reduced and is deposited upon the surface. After a few moments absorb this solution and repeat the process in order that more silver may be reduced and deposited.

"It is well to protect the hands with rubber gloves or to wear finger-cots. Throughout the treatment the silver that is deposited in the dentin is black. It is no longer silver nitrate; it is no longer formaldehyd; it is simply metallic silver that is deposited, with the formation of weak formic acid, which latter is readily converted into carbon dioxid and water. Sound dentinal tissue does not discolor, but any carious tissue appears jet black. Any tooth, even an anterior one, can be protected from the discoloring effects of these solutions by a coating of adhesive wax. By neatly cutting away the wax the treatment can then be applied at the point where it is indicated."

Rickert has modified Howe's method and he employes the following procedure: "The silver solution used for rapid reduction is made up as follows: Silver oxid is precipitated from a silver nitrate solution by KOH or NaOH. This is carefully washed to remove all impurities and kept moist in a small amber-colored bottle. In this condition reduction is so slight that we have kept it for a long time without much change. If a small amount is insoluble in excess of ammonia there has been too much reduction and the silver oxid should be freshly prepared. This is our stock solution made from silver nitrate, because it is free from nitric acid and other impurities.

"Now when we desire rapid reduction, as in a pulp-capping or root reaction, the silver oxid is added to a drop or two of ammonium hydroxid to the point of saturation. In this state the ammoniacal solution is easily reduced. The ease with which reduction takes place with this solution is so marked that even burnishing with a warm glass rod is sufficient to reduce it to the lustrous metallic silver. There is one precaution that must be mentioned here. That is, that after a few hours, fulminate of silver may be formed from the ammoniacal solution, which is very explosive. I have taken old solutions of both Dr. Howe's and my formulas and incompletely reduced the same in teeth or on blotting paper, both producing explosions on being touched with a hot platinum wire or by use of the electric current. Serious accidents are possible from

even wet solutions. Only very small amounts of ammoniacal solutions should be made and the unused portion discarded immediately after treatment. After complete reduction there is no danger and only old ammoniacal solutions, especially after drying, are to be avoided."

The application of silver nitrate for the purpose of root-canal sterilization possess certain advantages, which, however, are completely nullified by its numerous disadvantages. It is true, as we have stated, that silver nitrate is the best sterilizing medium of such infected dentin which for some reason cannot be excavated. However, if the employed ammoniacal solution passes beyond the foramen in the treatment of infected root canals, it is almost sure to cause severe pericementitis. A simple concentrated solution in distilled water is less irritating; it, however, as well as the ammoniacal solution will at once produce a solid precipitate upon the surface of the soft tissues which prevents its further penetration into the tissues and, consequently, deep infections of the tissue are not reached. Only a very diluted solution, *i. e.*, about 2 per cent will allow a fair penetration into deeper structures. The greatest objection to the use of concentrated silver solution consists in the permanent deep black discoloration of the root which is so very pronounced in cases of anterior teeth that often it may be visible through a thin alveolar process and anemic gum tissue. A bluish-black colored outline of the root upon the gum surface is the unsightly result.[1]

[1] For a more detailed discussion, the reader is referred to Prinz: Materia Medica and Therapeutics, 5th ed., St. Louis, Mo.

CHAPTER XVIII.

REINFECTION OF ROOT CANALS.

If a primarily infected root canal which has been treated by approved methods and thereby rendered sterile upon its surface is sealed with cement and with an antiseptic dressing left *in situ* for some weeks, it will be found that at a subsequent bacteriologic examination the canal again is, in most instances, reinfected with precisely the same organisms which caused the original infection. These reinfections apparently occur in chronologic sequence. Mayrhofer has shown that at the end of the first week after complete surface sterilization about 75 per cent of the treated canals proved to be sterile; at the end of the second week about half and at the end of the eighth week only 10 per cent remained sterile. The nature of the employed antiseptic, *i. e.*, in this particular instance formo-cresol, seems to make very little difference, although we have observed that electro-sterilization or the application of dichloramin-T slightly lengthens the period of temporary sterility. However, one important fact stands out preëminently: *An incipiently infected root canal cannot be sterilized permanently by the antiseptic methods of treatment now in vogue.* To completely sterilize an infected tooth root, basing our conception upon our present knowledge of antiseptic action, means to remove the tooth bodily and thoroughly boil it. Irrespective of this established fact, contrary statements are commonly found in current literature. Infectious material may be obtained at almost any time from a so-called sterilized tooth root and when placed in a nutrient medium will always show a luxuriant growth of pathogenic microörganisms. Complete sterilization of infected tissues can only be attained by the harmonious coöperation of three important factors: Absolute contact, time during which this contact is maintained, and sufficient concentration of the employed antiseptic. A break in the circuit always indicates failure. The complicated system of dentinal tubules which traverses the dentin from the pulp canal toward the periphery offers ready hiding places to the infective organisms and as the interior of the dentinal tubules cannot be reached by mere surface application of an antiseptic, the destruction of these deep-seated bacteria will always be a failure. These very conceptions are clearly set forth by the late W. D. Miller as early as 1890 when he stated that: "We must, in the first place, reconcile ourselves to

the fact that a complete sterilization of a suspected tooth with the conservation of the periosteum is absolutely out of the question."

The most momentous question arises at once: How does this reinfection occur? Four possible modes suggest themselves. The reinfection may start by gaining entrance into the filled canal from the pulp chamber; it may occur *via* the circulation; it may pass along the pericementum from the gingival margin, or it may take place from organisms left in the canal or in the dentinal tubules. The first route is rather difficult to imagine, especially when the root canal filling, which is naturally more perfect near the pulp chamber, is covered by a layer of oxychlorid cement. This cement offers an effective barrier against reinfection. Secondary invasions from areas about the empty apical region of pulpless teeth through hematogenous systemic infection do occur in the same manner as, reversely, they are observed in other organs of the body from focal infections. Reinfections from a marginal pericementitis which by continuity spreads toward the apex does also occur. Such cases are comparatively rare and are easily diagnosed.

The greatest majority of reinfections may be explained upon the basis that the infective organisms have remained undisturbed within the body of the tooth structure and they have escaped destruction during the supposed process of sterilization. It should be remembered that one solitary organism left unharmed may be responsible for the entire reinfection as the power of self-propagation of bacteria is enormous. As the late Miller has estimated, one single cell may produce 16,000,000 offsprings within twenty-four hours. The unharmed organisms must, of necessity, have found a safe harbor within the complex system of dentinal tubules which traverse the dentin everywhere or in the finer deltoid canals branching off from the main root canal near the foramen. By an ingenious method of counting Roemer succeeded in estimating the number of these tubules. Near the amelo-dentinal junction he counted some 14,700 tubuli within the area of a square millimeter. The number of these tubules progressively increases in the direction of the apical foramen in which he found 37,600 tubules per sq. mm. The width of these tubules, according to Koelliker, may be estimated as being from 1.5 to 4.5 μ, while the average size of a streptococcus is about 1 micro-millimeter, hence we readily understand how these organisms find suitable hiding places within the tubules. The root canals at their terminal ends near the foramen, as stated above, shows marked deviations. Hess has found that in 25 to 80 per cent of cases in a very large number of examined teeth of all types of the human denture from 2 to 5, and some times even more, additional foramina could be observed.

From the clinically observed facts it is evident that the exhaustion of any medicament sealed into the root canal occurs within a

day or two. The interior of the dentinal tubules cannot be reached by the superficially applied antiseptic as no direct contact is secured, hence the quick self-propagation of the retained infective organisms which grow luxuriantly into the empty or ill-filled root canal. It is self-evident, therefore, that the complete mechanical occlusion of the root canal in its entirety offers the only absolute guarantee for safety as regards reinfection. The complete corking-up of every tubule and of every accessory foramen is the final desideratum we must strive for in the permanent filling of a root canal and thereby deposit, as it were, an antiseptic which mechanically occludes the confined infective organisms and prevents renewal of their growth. The usual procedures at present in vogue with many practitioners

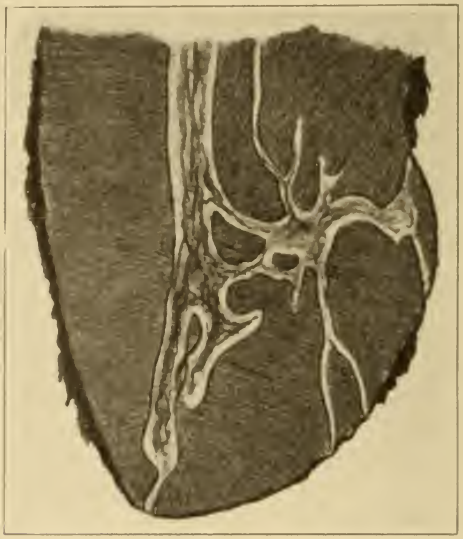


Fig. 134.—Arborization of the pulp of an upper second premolar near the apical foramen. (Fischer.)

is to leave doubtful canals unfilled for some time in anticipation of some future disturbance. Such unfilled canals, as experimental and clinical evidence clearly indicates, usually become reinfected within a week or two. Hence our tenacious adhering to this orthodox doctrine: An incipiently infected root canal of a tooth *in situ* may be rendered aseptic upon its surface by the use of certain antiseptic procedures, but it is impossible to sterilize the deep-seated infected tooth structure. As reinfection is primarily caused by organisms left undisturbed in the dentinal tubules, it should be an imperative rule with the operator to fill at once as completely as possible every root canal which has been found sterile upon its surface at the last visit of the patient.

In connection with this most interesting problem of reinfection of root canals, the question has often been asked: Is it possible for microörganisms or drugs to pass through the dentinal tubules, through the cementum into the pericementum? The significance of such a possibility is naturally of the utmost importance relative to the future welfare of the respective teeth. In a recent communication by Hopewell-Smith[1] we find the following authorative statement directly bearing on this subject:

"Attention must be directed for a moment to the homogeneous layer of dentin. Immediately external to the granular layer of Tomes, found in the dentin of the roots of human teeth, comes this important layer. It is an exceedingly narrow strip of homogeneous tissue, which is entirely devoid of any histological elements whatsoever. It is bounded externally by the first formed cementum, internally by the granular layer of Tomes. Its importance will be readily conceded when it is pointed out that this homogeneous layer acts, by virtue of its position and its structureless character, as an efficient barrier to the outward passage of drugs or medicaments placed in the root canal in the treatment of pulpless teeth. Assuming that drugs can travel to the utmost extent of the dentinal tubes—and it is to be doubted—it is impossible in normal conditions for them to pass into the cementum and so to the periodontal membrane. Thus, fortunately for mankind, one may use indiscriminately, and without fear of injuring the cementum or periodontal membrane, any or every drug of an escharotic or other nature. Otherwise, there would always be the possibility of the production of inflammation of the periodontal membrane, with subsequent loosening and loss of the teeth."

Bacteriologic Examination of Treated Root Canals.—Before a primary infected root canal which has been treated by anyone of the conservative methods is ready for the final filling it is imperative that its sterility should be established. This examination should not be made until the clinical evidence warrants the procedure. In regard to the microscopic examination of the treated canal it must be clearly kept in mind that the examination of a smear is less exact and delicate than the cultural control would be. Furthermore, it is necessary to realize that the method given below merely indicates when *not to* fill a canal and it does not furnish positive information regarding the sterility of the latter. Finding bacteria in the smear distinctly contraindicates filling of the canal while observing their absence does not mean that the canal is absolutely sterile. Cultural controls of treated canals which should be incubated for at least seventy-two hours will positively establish whether a root canal is sterile or not.

[1] Journal of Dental Research, 1920, No. 1, vol. **2.**

Microscopic Examination of Root-canal Smears.—1. Smear widely and evenly over a clean slide the dressing obtained from a root canal immediately upon its removal.

2. Dry smear in air.

3. Fix by passing rapidly through flame three times.

4. Stain for three to five minutes with Löffler's alkaline methylene-blue solution.

5. Wash off stain with tap water.

6. Invert cover-glass on slide film side down.

7. Blot dry.

8. Place drop of cedar oil upon stained film.

9. Examine with oil immersion lens (1.9 or $\frac{1}{12}$ inch).

CHAPTER XIX.

FILLING OF ROOT CANALS.

From a perusal of the early records of the practice of conservative dentistry, *i. e.*, the works of Fauchard (1728), Bourdet (1757), Hunter (1778), and others, we are informed that whenever a "nerve" in a tooth was exposed and diseased, it was destroyed by the actual cautery or by a potential caustic, which, however, as the keenly observing John Hunter remarks "is not always possible." These early writers also referred to the medicinal treatment of the "fangs;" the French dentists being especially partial to the use of "essence de giroffle" (oil of clove) and Hunter prophetically emphasized antiseptic procedures of a somewhat similar nature as being essential for the future preservation of the afflicted tooth. Cotton-wool, moistened with oil of clove or some other antiseptic compound, was usually sealed into the root canal prior to the final filling of the tooth cavity.

The use of soft metals for the filling of root canals, *i. e.*, gold, lead or tin, was instituted later. It is quite certain that the better class of American dentists practised root-canal filling at the beginning of the nineteenth century. In 1826 the international Koeker stated that "the practice of dentistry has reached a state of development in the United States which is higher than in any other part of the world, although very little is published by American dentists." Fortunately, certain proofs are available to the student of dental history which makes it plain that root-canal filling must have been a well-known procedure to the high-class practitioner in those days, for we find among other data a bill rendered in 1824 by Edward Hudson, a prominent Irish dentist, who practised in Philadelphia from 1805 to 1833, in which he itemizes "stopping the cavity of one tooth from the end of its root with gold." The latter material was in common use for such work up to the early seventies of the last century and such men as Maynard, Barker, Dunning, Forster, Westcott, Palmer, Evans, McKellops and a host of others, were strong advocates of the special fitness of this metal for the above purposes. In 1849 the once famous Hill's stopping was introduced as a general filling material and it was also advocated as a suitable root-filling compound. It was superseded, in 1872, by a solution of gutta-percha base plate in chloroform, which, however, was quickly modified by employing a solid cone of this same material in conjunction

with this solution. As a close second, oxychlorid of zinc cement, in the early days better known as Houghton's or Robert's os artificial, was highly prized by many operators as being the most suitable material for the permanent obliteration of root canals. From this collection of historical data it is apparent that the closely observing practitioners of the past were aware of the fact that a root canal deprived of its natural contents, *i. e.*, the pulp, must be filled with some solid substance in order to prevent subsequent infection.

The primary object of filling a root canal is to replace as perfectly as possible the artificially or pathologically destroyed pulp with a solid inert and unchangeable substitute. The specific properties of the substances used for such purposes should possess the following qualifications:

1. The material must be non-putrefactive.
2. It must be sufficiently plastic to allow its easy introduction and adaptation to all parts of the canal.
3. It must not change its form after being inserted.
4. It must be impervious to moisture.
5. It must seal the dentinal tubules and the apical foramina hermetically against bacterial invasion.
6. It must not discolor the tooth substance.
7. It must not irritate the soft structures beyond the foramen.
8. It must, in case of necessity, be easy of removal.
9. It must offer resistance to the roentgen rays so as to furnish an opaque shadow picture.

To enumerate all the substances which have been advocated at one time or another as root-filling materials and to criticise their respective usefulness is quite impossible at this moment, as a consideration of all the merits and demerits of each compound would comprise a small volume in itself. Merely to mention the most important groups we may state that silk or cotton, either raw or purified, medicated or carbonized, asbestos, the cements, metals in the form of wire, foil, or as amalgam, gutta percha in its pure form or in its many modifications, wood points, semisolid pastes principally composed of zinc oxid combined with various antiseptics; balsams, resins, waxes, liquid bakelite, etc., have been used with more or less success. Clinical observations and laboratory experiments have amply demonstrated the fact that out of this great mass of suggested materials only a very few substances answer the above demands.

Silk and cotton in its pure state or in its many modifications have always been a failure as a root-filling material. In the few cases in which cotton fillings have proven to be successful, it must be assumed that the chances for infection were negative or the sterile cotton plug near the apex was sufficiently solid to prevent the passage of bacteria. Among the various cements, the oxychlorid

of zinc has been highly lauded and such skilled operators as Jenkins, Kells, Ottolengui and others recommended it as being practically the most suitable material for this purpose. Zinc oxychlorid cement if forced beyond the foramen is markedly irritating and its complete removal from a root canal in cases of necessity is often impossible. Metals, on account of their imperfect adaptation and natural resistance to manipulation in the minute canals have been practically discarded. Gutta percha, in the form of cones, in conjunction with its solution in chloroform, the essential oils or some other vehicle and covered with zinc oxychlorid cement, as Webster and Cook have shown experimentally are practically moisture proof, and prevent the ingress of bacteria. Cook was rather partial to a chloro-percha solution, claiming that a slow liberation of active chlorin, which acts as a powerful antiseptic, takes place in a root canal filled with this compound, while other practitioners prefer a solution of gutta percha in eucalyptol or in any one of its isomers, *i. e.*, cajuputol, myrtol, cineol, etc. Laboratory tests made with these compounds in glass tubes and in extracted teeth have demonstrated that a water-tight filling can be made. If sterile gutta percha is accidentally pushed beyond the foramen without injuring the soft tissues it will be readily tolerated. In an aseptic area it is encapsulated like the proverbial "lead ball." Gutta percha is a non-putrefactive substance, sufficiently plastic to allow its ready introduction and it will not change its form after being packed against solid walls. It may be readily removed if need be, and it does not discolor the dentin. In addition, it offers marked assistance to the passage of the roentgen rays. In general, it may be categorically stated that gutta percha is the most satisfactory material for the purpose in view, as it agrees in every respect with the above enumerated requirement of a suitable root-filling compound. This statement is amply supported by clinical experience as gathered from observations covering more than half a century. The principle causes of the recorded failures may be attributed to an inaccurate technic of its manipulation and to improper preparation and treatment of root canals.

Gutta percha in its various modifications is principally employed in the United States and Canada as a root-filling material, while in Europe semisolid pastes are still largely employed for such purposes. Experimental work with the latter compounds has proved that they are valueless; they prevent the ingress of sepage only temporarily. Wooden points, either artificially prepared or in the form of natural thorns or as charcoal or fiber points, are porous and consequently are useless substances. Salol, when melted into a root canal, will be absorbed within a short time. Some of the balsams, *i. e.*, Canada balsam, balsam of Peru, balsamo del Deserto, concentrated solutions of sandarac and other resinous

compounds, are still lauded by many practitioners. The solid resins possess many advantages over other compounds; their successful introduction into fine root canals is very difficult. In very thin solutions, however, they are most excellent adjuncts. Within the last decenniums high fusing paraffin has been favorably recommended as possessing all the enumerated qualities. It has been claimed that the paraffin is absorbed from root canals and, consequently, infection of the empty canal may occur at some future period. The writer is under the impression that in most instances the spaces occurring from supposed absorption have never been correctly filled or that a low-fusing paraffin had been employed. Satisfactory roentgenologic controls of ten and more years' service of root canals filled with hard paraffin containing 30 per cent bismuth oxid are sufficient proof of this assertion.

FIG. 135.—Gutta-percha cones.

FIG. 136.—Hard paraffin root canal filling. The roots were filled fourteen years ago with hard paraffin-bismuth compound and the tooth is still in position on May 2, 1921. (Dr. Arch. Miller.)

The addition of antiseptics to insoluble substances, *i. e.*, gutta percha, cements, etc., which are used as root-filling materials has practically very little value. Theoretically, antiseptic action indicates that the substances must become soluble, or to speak more correctly the pharmacologic action of a drug is manifested only when the drug enters the tissues as a liquid or in gas form. When a solid drug is introduced it necessarily follows that it must be soluble in the tissue fluids if its typical action is to be manifested. Since gutta percha and other similar substances are insoluble in the body juices, they prevent the solution of the drug or drugs which are dissolved in or incorporated with them. The insoluble porous root-filling material, *i. e.*, cotton, wooden points, various cements, semisolid pastes, etc., containing a soluble antiseptic offers no resistance to the invading surrounding fluids; it will be exhausted in due time. This statement is sufficiently substantiated by clinical observation and experimental work. Inocu-

lated plate cultures in which cones of sterile gutta percha containing reasonable quantities of thymol, mercury bichlorid, bismuth oxid, iodoform, etc., are suspended, show a more or less tiny zone of inhibition of bacterial growth within their immediate periphery. It is an indication that some of the antiseptic material deposited upon their surface has entered into solution. But these small quantities of the antiseptic are too minute to be regarded as being of permanent value. No bacterial growth is observed upon suspended pure sterile gutta-percha cones. If we wish to cover a root canal with an antiseptic it seems more reasonable to apply this antiseptic as such or dissolved in a volatile medium prior to the insertion of the final solid canal filling. Of the many antiseptics which, relatively, possess permanent action, comparatively few answer our purpose. A normally liquid antiseptic is not as well suited as one which can be deposited in dry form and which slowly dissolves in the moisture present in the dentinal tubules. It should be borne in mind that no substance will enter the dentinal tubules of a tooth root *in situ* at once, on account of the present moisture; it requires a more or less prolonged period of time (days and weeks) to accomplish this process and it should also be remembered that experiments regarding the penetrability of antiseptics made with teeth out of the mouth do not represent normal conditions and are, therefore, misleading. From experimental work we are led to believe that aceton is a fluid which is especially suited as a carrying medium of antiseptics on account of its ready affinity for water, its solvent action upon fatty substances, its penetrating power, its hardening effect on tissues and its ready evaporization. Aceton is a colorless mobile and volatile liquid having a characteristic ethereal odor and a pungent sweetish taste. It volatilizes at a low temperature and boils at about 133° F. (56° C.). It is readily soluble in water, alcohol, ether, chloroform and the volatile oils.

We again emphasize that, according to our conception, the object of a root-canal filling consists in sealing hermetically the foramina and the tubules with a solid, inert and unchangeable substance. The *complete* obliteration of the canal is the only logical solution of this problem. The sealing of the dentinal tubules is of paramount importance since reinfection of a root canal is probably always due to the renewed activity of those microörganisms which lodge within the dentinal tubules and which cannot be destroyed by surface disinfection.

At present it is generally conceeded that the solid obliteration of a sterile empty root canal with gutta percha, from a clinical point of view, offers serviceable chances against reinfection. The difficulties encountered in obtaining the desired results rest primarily with the mechanism involved in the complete occlusion of small

tortuous canals and *not* with the material. Hence the important dictum: Every root canal must be, if possible, mechanically enlarged to its very apex. To facilitate the ready introduction of gutta percha, its solution in various solvents, especially in chloroform or eucalyptol, have been advocated. It is a well-known fact, however, that such solutions on the evaporation or resorption of the solvent must naturally shrink. The empty space produced by the shrinkage leads, as we have stated above, to reinfection. On the other hand, it is also proven that when such solutions are used in minute quantities merely as lubricants for the gutta percha cone and by applying sufficient force in the final packing a perfect adaptation of the semiplastic gutta percha is obtained so as to produce an absolute water-tight filling. The clinical success obtained with this procedure in the hands of expert operators is ample proof of the soundness of this statement. It must be admitted that not every operator possesses this superior dexterity which, however, must not be construed as shielding slovenly work in regard to the too liberal use of gutta-percha solutions as well as insufficient care in the final packing.

To further facilitate ready obliteration of minute spaces left by an ill-adapted gutta-percha cone, the late Callahan, in 1910, introduced a most ingenious combination of root-canal technic consisting of the use of a very thin resinous solution in conjunction with the usual gutta-percha cones. Callahan stated in his diverse writing: It required a long time for me to realize the advantage in the use of a thin solution. A thick mixture will not penetrate the tubules nor does it give up enough chloroform to dissolve gutta percha. The use of a *very thin* rosin solution is the most essential feature of his discovery. However, it should be borne in mind that if one depends upon the solvent action of the chloroform upon gutta percha the evaporation of the solvent naturally produces shrinkage and hence, an empty space between the cone and the wall of the canal results. If, on the other hand, the thin rosin solution is allowed to percolate into the previous dehydrated dentin and the solvent is evaporated prior to the final filling of the canal, no shrinkage will occur.

Callahan advocated the following solution for such purposes:

French violin rosin	12 grains (0.75 gm.)
Chloroform	3 drams (12.00 gm.)

Neither the nature of the rosin or its solvents are essential features of the solution. Any good rosin and a suitable solvent of a very low boiling-point answer the purpose equally well. The writer prefers a good, clear rosin dissolved in aceton. If a neutral solution of the normally slightly acid rosin is desired a few grains of sodium bicarbonate may be added. After the dehydrated dentin

is saturated with this thin rosin solution and the solvent has evaporated, the dry glazed interior of the canal is coated with a very thin film of a suitable lubricant to facilitate the ready placing of the final gutta-percha cone. A solution of gutta-percha base plate in paraffin oil, equal parts, is most serviceable for this purpose. This solution does not shrink as it will not evaporate. Again it should be emphasized, that this paste merely acts as a lubricant and that only the smallest possible quantity should be employed.

The rationale of this method of filling root canals may be explained upon the following basis: The dehydrated dentin offers good chances for the ready percolation of the very thin rosin solution into the dentinal tubules to the depth of a few millimeters which thereby are corked up. In addition, the canal itself is tightly obliterated by a solid packing of gutta percha against a sterile glazed surface.

In using a solid cone for filling a root canal the question is often asked: What becomes of the confined air? This little air when forced beyond the foramen will be taken care of by the soft tissues and since it is not forced into a vessel, no danger from air embolism is to be expected. That such small quantities of air, even if injected into a vessel, are practically free from danger, has been experimentally demonstrated by Blair and McGuigan.

Experimental work carried out on freshly extracted teeth shows that the mechanism of dentin infiltration, etc., as outlined above actually occurs. If the rosin solution is colored with Sudan red, ground stained sections of the root show its deep penetration into the dentin. If the teeth, whose root canals have been filled according to the outlined method, are immersed in methylene-blue solution and by a glass tube sealed into its pulp chamber are connected with a suction pump under water pressure, it will be found that an absolute water-tight, root-canal filling can readily be obtained.

Technic of Filling Root Canals.—A systematic analysis of the various steps involved in the practice of filling root canals as based upon our previous discussion in regard to reinfection, resolves itself into the following procedures: Dehydration of the sterile root canal, infiltration of the dentinal tubuli with a very thin rosin solution, evaporation of the rosin solvent, coating of the varnished canal walls with a thin film of gutta-percha paste, the final solid plugging with pieces of gutta percha and filling of the pulp chamber with zinc oxychlorid cement. A final roentgenogram is essential for checking-up the operation.

Dehydration.—The *sine qua non* of a successful root-canal filling is an absolutely dry root canal. To accomplish this end, certain physical procedures are in vogue, *i. e.*, the hot air blast, the electrically heated root dryer, the heated wire, of which the Evans' root dryer probably is the best known prototype, bibulous paper cones,

Fig. 137.—Evans' root dryer.

cotton, etc. To facilitate the removal of moisture, hygroscopic chemicals, *i. e.*, alcohol, chloroform, ether aceton and other substances, are often used in conjunction with the above enumerated means. These compounds, with the exception of aceton and alcohol, have little affinity for water and hence are of no practical value. As we have stated above, aceton or absolute alcohol is admirably suited for this purpose. In drying out a root canal it should be borne in mind, that the removal of its natural moisture or any other fluid placed into it is well-nigh impossible with the much lauded hot-air blast if the foramen is closed. A few trials on an extracted tooth or a glass tube drawn out to a fine solid point and filled with water or any other of the above enumerated fluids will readily convince one of this illusoric conception. The fluid will move back and forth upon the elastic cushion of air confined in the end of the tooth or the tube, or, if no air is present the

Fig. 138.—Riethmüller root dryer.

heated air blast will practially make no impression on the moisture column. The removal of moisture is usually best accomplished by using bibulous paper cones in conjunction with a heated metallic root-canal dryer. The cessation of the hissing sound following the introduction of the hot wire indicates that the desired effect has been successfully achieved. In passing, it is well to remember that over-drying the tooth structure is a dangerous procedure. If more or less of the water which holds the gelatinous matrix of the tooth in colloidal solution is removed by over-heating, that tooth is proportionally weakened against physical or chemical insults, a fact which is well borne out by clinical observation.

Infiltration of the Dentinal Tubuli with a Thin Rosin Solution.—The solution required for this purpose consists of:

Rosin	15 grains (1.00 gm.)
Sodium bicarbonate	4 grains (0.25 gm.)
Aceton	2 ounce (15.00 gm.)

Decant the clear solution and keep in a well-stoppered bottle.

With a paper cone the solution is pumped into the root canal immediately after dehydration and the aceton is evaporated with the warm air blast. If necessary, the process is repeated so as to have a fair assurance that every tubule is sealed.

Coating the Canal with a Gutta-percha Paste.—A most suitable paste for this purpose which will not shrink is prepared as follows: ½ ounce of gutta-percha base plate cut into small pieces is put into a suitable perfectly dry wide-mouth bottle and covered with ½ ounce of a colorless pure paraffin oil (nujol, etc.). The bottle containing the mixture is placed on a heated sand bath until complete solution is obtained. On cooling, the mixture has the consistence of butter in cold weather. With a broach a very small quantity of this paste is transferred to the varnished canal and spread over its wall. A blast of warm air will greatly assist in its even distribution. The object of using this paste is primarily to place a lubricant into the canal to facilitate the ready adaptation of the gutta percha under pressure, and secondarily to occlude every nook and corner by an unshrinkable material. We again emphasize that *only the smallest possible quantity of this paste must be employed.*

Final Plugging of the Root Canal.—Before starting the final operation of plugging the root canal the roentgenogram with the diagnostic wire in position is carefully examined so as to obtain a fair

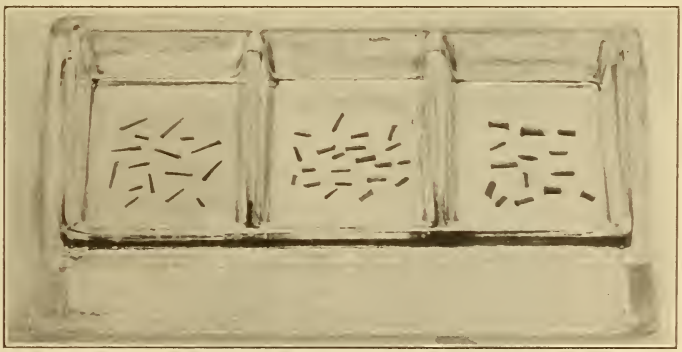


Fig. 139.—Glass contained for gutta-percha cones. Gutta-percha cones cut in short lengths ready for use. (Coolidge.)

conception of the size, general outline and length of the canal. To approximately determine the size of the foramen, a suitable sterile root-canal plugger is selected and inserted. Too small a plugger will pass through the foramen which is indicated by pain. The correct instrument should pass up to, but not quite to the foramen. A very small piece of rubber dam stretched over a smooth broach may be used as a guide to determine the length of the canal. A suitable sterile gutta-percha cone which approximately fits the lumen of the canal is selected from the cone sterilizer and is cut

into pieces about $\frac{1}{8}$ inch (3 mm.) in length. A suitable piece which approximately will fit the upper end of the canal as determined by the plugger point is selected, mounted upon the warmed end of the sterile canal plugger and carried to the canal. The gutta percha must never be touched with the fingers. The piece is slowly but very firmly pressed to place. Slow, firm and persistent pressure is imperative so as to drive the piece, if possible,

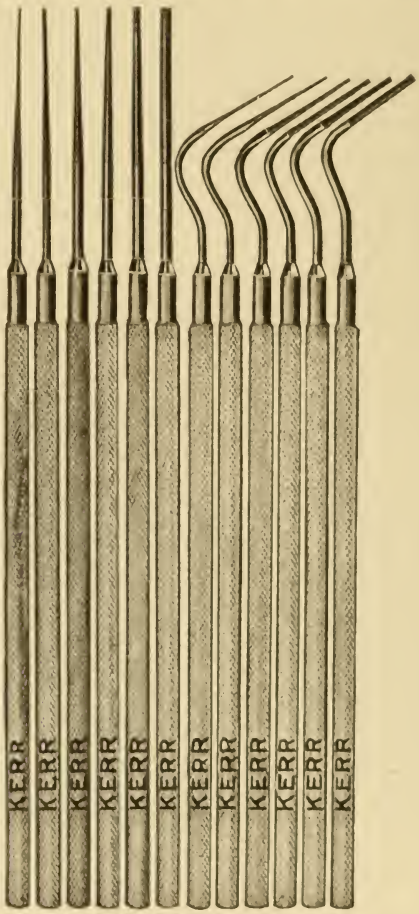


FIG. 140.—Root canal pluggers.

to the very apex or even slightly beyond it. Under the persistent firm pressure the semiplastic gutta percha will adapt itself to the irregularities of this region. The placing of the initial piece of gutta percha is the most important part of the entire operation of filling a root canal and not too much emphasis can be put upon carrying out its correct technic so as to obtain the desired results. The hermetical sealing of the foramen without injury to the peri-

apical tissues is the unalterable prerequisite upon which the future welfare of the tooth rests. Clinical experience has amply demonstrated the fact that this small quantity of sterile gutta percha which accidentally may pass beyond the foramen is too insignificant to be productive of serious consequences. The remaining portion of the canal is solidly filled in the same manner with gutta percha bits as needed, using larger root-canal plugger and the pulp chamber is finally occluded with oxychlorid of zinc cement. A roentgenogram may now be taken so as to check up the operation. If the root-canal filling is in any way defective it should be removed. The application of chloroform or xylol to the gutta percha assist in softening the cone and thereby facilitate its removal. The canal should be refilled at once as outlined above and a second roentgenogram is taken to show whether the operation is a success or failure.

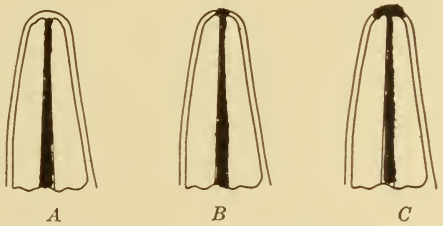


FIG. 141.—*A*, filled to the end; *B*, crater filled; *C*, apex capped. (Crane.)

Summary of Filling Root Canals.—1. Carefully examine the roentgenograph taken with diagnostic wire in position prior to filling the canal in regard to its length, size and general outline. Determine the size of the foramen with a root-canal plugger and the length of the canal with a smooth broach having a small piece of rubber dam attached to serve as a guide.

2. The suitably enlarged canal is dried with sterile paper points, followed by aceton or absolute alcohol and the hot root dryer.

3. Infiltrate the dentin with the rosin-aceton solution carried upon a paper cone and dry with warm air.

4. Apply a *very small* quantity of gutta-percha-paraffin oil paste on a broach. A touch of warm air will facilitate its even distribution.

5. Select a sterile gutta-percha cone and cut it into pieces about $\frac{1}{8}$ inch in length. Select a suitable piece, mount it upon the warmed sterile root-canal plugger and carry it to the canal. Do not touch the gutta-percha cone with the finger. Apply slow but very firm and persistent pressure. Fill the remaining portion of the canal with gutta percha bits in the same manner, using larger canal pluggers as needed.

6. Fill the pulp chamber with oxychlorid of zinc cement.

7. Have a roentgenogram taken to check up the operation.

CHAPTER XX.

ACCIDENTS ARISING IN THE TREATMENT OF ROOT CANALS.

I. THE BREAKING-OFF OF INSTRUMENTS IN ROOT CANALS.

In the process of mechanically cleansing a root canal of its contents it occasionally happens that a broach or other steel instrument will break and remain lodged in the canal. An accident of this type should not always be attributed to the awkwardness of the practitioner; it may occur at any time even in the hands of the most expert operator. On the other hand, it should be stated that certain precautionary measures may obviate, or at least materially reduce the possibilities of such accidents. In general, one should be mindful of the fact that the principal danger of breakage lies within the very delicate nature of the instruments as utilized for the purpose in view. As a consequence, it behooves us to purchase only the very best quality of root canal instruments and to carefully test them in regard to temper, flexibility, etc., prior to their employment.

To further obviate the possibility of breakage the use of engine-driven root-canal instruments of a delicate type, *i. e.*, Gates-Glidden drills, etc., should be discouraged; they are largely and correctly supplanted at present by improved hand instruments, especially files, etc. Of the various hand instruments, aside from broaches, the root-canal reamers furnish the largest quota of breaks. The very nature of their construction, being in reality modified forms of twist drills, favors their ready fracture. It is claimed by Clawsen that if the broach is inserted and held in such a manner that pressure may be produced upon the broach toward the apex of the root, so that the stem of the broach is free from all edges of the cavity and not allowed to rest against any edge during the rotation, the broach may be rotated at will, reducing the danger of breaking by at least 50 per cent. In removing the broach, slide the finger to the opening of the cavity and raise the broach with the finger at that point, thus avoiding any binding upon the edges of the cavity, and rendering the removal of the broach very easy and sure.

The various methods at our command for the removal of broken instruments from root canals may conveniently be divided into mechanical measures, chemical methods and surgical procedures.

Mechanical measures intent to remove the broken part by manipulative skill and the various methods utilized for such purposes should always be employed at first in preference to any other procedure. If the broken piece can be grasped and removed with suitably shaped delicate pliers, of course, the incident is closed. If, on the other hand, the attempt fails, additional means may be called to assist in the effort. A very few fibers of cotton may be loosely wound about the finest barbed broach and introduced into the canal alongside the broken piece. By turning it to the left, the piece may become entangled in the cotton and thus its removal is occasionally successfully accomplished. William H. Trueman described the following device for such purposes: Taking a piece of fine brass wire, such as is used to keep open the needle of a hypodermic syringe, he formed upon one end a spiral of a few turns by winding it upon an instrument about the same size as the broken one and securing the other end to any small tool with gum shellac. This spiral he placed over the broken instrument in the pulp canal, pushing it well down with a broach. Upon gently withdrawing it, the coils of the spiral tightened upon the broken instrument, holding sufficiently firm to effect its removal.

Trueman's device furnished the prototype for the so-called Beutelrock broken instrument

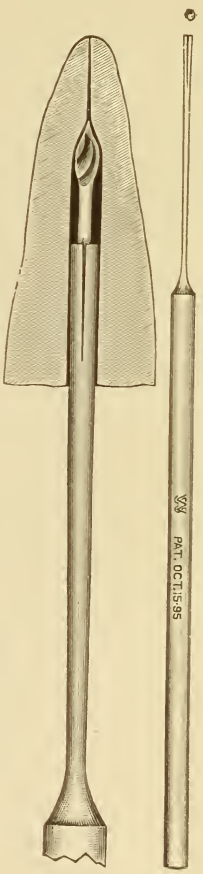


FIG. 143.—Split and threaded instrument for engaging the shank of a Gates-Glidden drill.

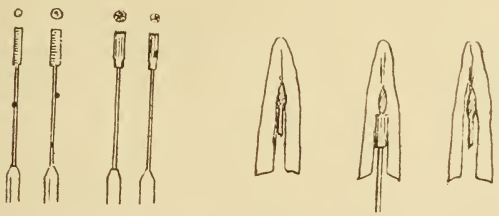


FIG. 142.—Beutelrock's instruments for the removal of broken broaches.

removers which may be obtained in various sizes. To facilitate the ready application of these wire spirals, small trephines are furnished with the Beutelrock set. A somewhat similar tool, especially designed to remove a broken Gates-Glidden drill has also been on the market. This extractor has a split and threaded cone socket, which, when carefully pushed and turned in the canal,

will seize the broken drill-shank and effect its extraction. In some cases it may be best to first enlarge the canal before inserting the extractor.

Crane suggests the following procedures: Broken instruments offer the greatest difficulty of any canal obstruction. It may be here pointed out, however, that this does not apply to smooth instruments broken off in the picking and pushing motion. If these are simply ignored for the time being and the picking and pushing resumed with a new instrument, the broken portion will soon ride out of the canal, but where an instrument is broken while being screwed into the canal further exploration is often impossible. By repeated use of sodium-potassium on a root pick, it is sometimes possible to make a pathway alongside of the obstruction, and then a twist broach may be passed into this and twisted around and around to the right, without permitting it to advance into the opening. When this does not work it is feasible occasionally to continue the picking alongside of the broken instrument and reënter the canal at a point apical to it.

Wass[1] employs an aqueous solution of iodin trichlorid and he gives the following direction for its application: "The 25 per cent aqueous solution is used for the purpose of dissolving broken-off steel instruments of any sort which may be found in root canals and which cannot be dislodged by mechanical means. Only tantalum- or platinum-pointed instruments or a platinum-pointed dropper or syringe should be used in applying the solution. The action of ICl_3 when it comes in contact with steel or iron is to form ferrous iodid and ferrous chlorid, both of which salts are soluble in water. Its action is definite and very rapid if sufficient surface of the metal is exposed, and may be tested by dropping a broach in a little of the solution. In a canal, however, it has only the cross-section of the broken metal to attack and it will often require more than one visit and patient technic to either reduce, say, a broken broach, sufficiently in circumference so that it will drop out of the canal, or to entirely dissolve it. Therefore, it is necessary to drill around the exposed end as much as is safe, so as to expose as much metallic surface to attack as possible. Apply a small quantity of the 25 per cent solution of trichlorid of iodin around the broach, in the canal and allow it to remain for three minutes. Absorb this with paper points, wash out with distilled water and dry around the broach with cotton wound on a broach, removing any remaining ferrous coating. Dry with air blast and pick around the instrument with Rhein picks. Apply ICl_3 again for three minutes; absorb, wash, dry, pick and enlarge the space around the instrument again, and repeat until the entire obstacle is removed. If

[1] Dental Cosmos, October, 1918, p. 808.

the tooth under treatment is an anterior tooth, where any discoloration must be avoided, the best procedure is to flow a small quantity of melted paraffin over the dentin which lines the labial enamel—in other words, the labial coronal dentin. This will not interfere with the action of the ICl_3 in the canal, and the crown of the tooth will not be stained. In posterior teeth, if there is any reason, remove the stain, it can be best accomplished by applying a strong aqueous solution of ammonia followed by a saturated aqueous solution of sodium hyposulphite."

Iodin trichlorid is a most powerful caustic which requires the greatest care in handling. From his experience with this compound, the writer is under the impression that the danger associated with its application for the purpose of dissolving broken-off broaches is too great to merit its general adaptation by the average practitioner.

Some years ago, while determining the solubility of dentin in the various mineral acids for the purpose of assisting in the opening of obliterated root canals, the writer[1] observed that the most satisfactory acid for such purposes is a 50 per cent nitric acid or a 50 per cent nitro-hydrochloric acid. Incidentally, it was observed that while the diluted nitric acid would rapidly dissolve a steel broach, the pure acid would do so only very incompletely, or, at least, very much slower. Furthermore, the peculiar fact became manifest, which however, as later inquiries revealed, was being known to chemists, that the solvent action of the respective dilution of the acid upon steel was restricted to very limited bounds. For example, pure nitric acid may require many hours to dissolve a piece of steel broach of a given weight and size, while a piece of the same character will completely dissolve in a 50 per cent solution of this acid within ten minutes and only slightly so in a 25 per cent solution. It should be understood that the term percentage solution as referred to these diluted acids indicates measures by volume. While experimenting with the various halogens for the purpose of sterilizing infected root canals by ionization, it was observed that when a steel needle was used in the presence of a concentrated Lugol's solution, invariably, an iron iodid or iodate was formed. In other words, the insoluble steel in the presence of liberated iodin was changed into a soluble salt. It was found that a few drops of a solution having the following composition will dissolve a piece of steel broach of average thickness and measuring $\frac{1}{4}$ inch in length within thirty minutes:

Potassium iodid	2 drams (8 gm.)
Distilled water	3 drams (12 gm.)
After complete solution add,	
Iodin crystals	2 drams (8 gm.)

[1] Prinz, H.: Dental Cosmos, 1922, p. 1105.

Keep in a glass-stoppered bottle. The bottle should be preferably kept in a tightly closed box and stored in the laboratory as the iodin fumes which may arise therefrom are highly destructive to steel instruments. The solution does not deteriorate with age.

It is necessary to realize the fact that a broken piece of steel as wedged in a root canal presents to the attacking solvent merely a cross-section of the broach, *i. e.*, an extremely small surface, hence the solvent requires a much longer time for its action. In addition, absolute contact between the steel and the solvent is an imperative necessity and while this most important fact is self evident, nevertheless, it is frequently overlooked and it was found to be the principal cause of recorded failures. To obtain as large a surface of exposure as possible, the preliminary application of diluted nitric acid to the root canal is strongly recommended. The acid naturally has a greater affinity for the less resistant dentin than for the steel; as a consequence, the dentin surrounding the piece of steel is readily decalcified. A drop of 50 per cent nitric acid when worked into a root canal with a platinized gold broach usually is completely neutralized in less than ten minutes. In clinical practice a drop of the diluted nitric acid should be worked into the canal about every two minutes until the canal is fairly filled. The acid is now neutralized with sodium dioxid carried on a barbed broach wound with a few fibers of asbestos and dipped in alcohol. The resultant evolution of oxygen often loosens the piece sufficiently that it may be washed out by a stream of water from a syringe. If, however, the piece is not dislodged, the concentrated iodin-potassium iodid solution as given above should now be applied. To prevent undue dilution of this solvent all the moisture has to be removed from the root canal. As the compound readily destroys steal, it is best applied with an eye dropper and worked into the canal with a platinized gold broach. Finally, a few fibers of asbestos are saturated with the solution and packed into the canal and the cavity is *carefully* sealed with temporary cement. The application should remain undisturbed for at least twenty-four hours. On return of the patient, the contents of the canal are washed out and with a fine barbed broach wound with a few fibers of cotton an effort is made to remove the débris. If the piece of broach is not completely destroyed a second or a third treatment may be applied as needed.

In spite of the various suggested mechanical and chemical methods, the removal of a broken broach from a root canal will not always be crowned with success. As a final desideratum, surgical procedures may be instituted. While in all cases of this character a good roentgen picture is most desirable; it is of imperative necessity for the surgical methods. The exact location of the broken piece will naturally guide the clinician in the specific technic

selected for the respective case. Williger[1] advocated excision of the root apex in such instances, and he proceeds as follows: Resection is most easily performed in upper anterior teeth, in which removal of a foreign body is most necessary. The tooth is opened far enough to allow the easy introduction of a straight root-canal plugger into the root canal. Under local anesthesia and anemia

FIG. 144.—Broken broach in root canal.

FIG. 145.—Broach removed by iodin-potassium-iodid solution and canal filled. (Dr. John Burkhardt.)

the root apex is exposed and resected as far as indicated, and the foreign body is pushed through the root canal with the root-canal plugger or a cut-off Miller broach in a broach-holder, until it can be grasped with pincers and extracted. The root canal is then cleaned with hydrogen dioxid and filled. If a broach has been in a canal for a long time it breaks easily in removal and must frequently be extracted in several pieces, together with a quantity

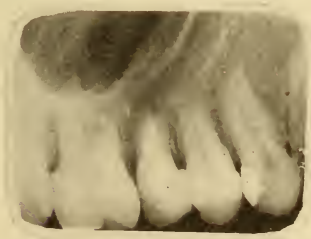


FIG. 146.—Broken broach above foramen.

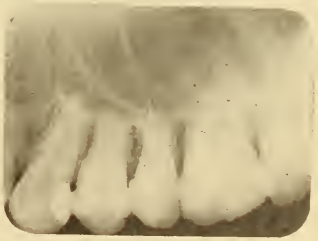


FIG. 147.—Appearance of tooth four weeks after the piece had been removed surgically by Dr. R. Foy.

of oxidized débris. In such cases the root shows dark blue discoloration. If no granuloma or cyst is noted after incision and exposure of the root apex, a shallow groove is burred in the bone in the longitudinal axis of the tooth with a strong, straight fissure bur. Through this groove the broach can be pushed out, grasped

[1] Dental Cosmos, December, 1912, p. 1289.

and extracted. In first and second molars this method is rarely applicable. Instead, the tooth is extracted, the foreign body removed and the tooth replanted under aseptic precautions.

II. PERFORATION OF A TOOTH ROOT.

Perforation of the root of a tooth may be brought about by an accident, *i. e.*, the misguided drill or reamer may pass through its wall during the process of opening up a canal or in excavating deep-seated caries the instrument may cause a parietal perforation through softened dentin. The accidental perforation should not always be attributed to the awkwardness of the operator. In the majority of cases the injudicious use of engine drills or hand instruments, especially by the inexperienced student, is probably most frequently responsible for such accidents.

In conformity with its position we may differentiate between a parietal and an interradicular perforation and according to its etiology, we may speak of a traumatic or pathologic perforation. While the parietal perforation which is usually the result of a trauma may occur anywhere between the pulp chamber and the apex of the root, the interradicular type is only observed within the pulp chamber of multi-rooted teeth and it is usually the sequence of some pathologic process, *i. e.*, caries. The passing of the broach through the apical foramen should not be designated as a perforation.

The subjective symptoms of a root perforation are usually rather definite, *i. e.*, the perforation itself is not painful, but on passing a probe through the opening into the pericementum, the patient feels a distinct painful sensation resembling the prick of a needle within the gum tissue. The character of the pain is distinctly different from that experienced in pulpitis or pericementitis and it becomes altered with the development of secondary infectious disturbances within the alveolus. Granulation tissue which may protrude through a pathologic perforation should be carefully differentiated from pulp tissue. A recent perforation is always accompanied by rather profuse hemorrhage.

If the perforation is situated in the pulp chamber or in the lower part of the root it may be frequently observed directly or by the aid of a mirror. The opening, on passing a broach, is distinctly felt by the guiding fingers and its withdrawal usually is followed by very free bleeding. In deeper perforations the diagnosis is usually wrought with difficulties. In such cases a roentgenogram with the diagnostic wire in position is an imperative necessity.

A differential diagnosis between a perforation and a pulpitis or a pericementitis is readily made by comparing the various symptoms as discussed under "General Principles of Diagnosis."

The prognosis of a perforated root depends entirely upon existing

conditions. If the perforation is of recent occurrence and located within an easily accessible region in a strong, sound root and if no suppurative infection of the pericementum is to be observed, the prognosis is rather favorable, while in opposing conditions the operator should not be too sanguine in his expectations. In the latter instances the successful closure of the opening always remains an operation of chance.

The treatment of a perforation must be based upon the possibility of establishing asepsis within the perforated wall and the surrounding tissues. While it is not necessary that all perforations should indicate actual sepsis, it is, nevertheless, important to look upon an exposed root canal as being infected. Whether the per-

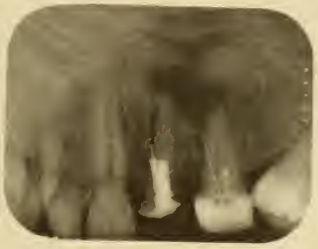


FIG. 148.—Two parietal perforations.

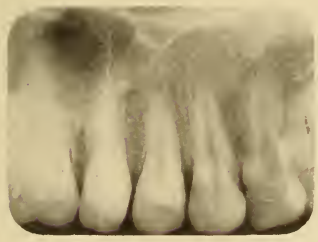


FIG. 149

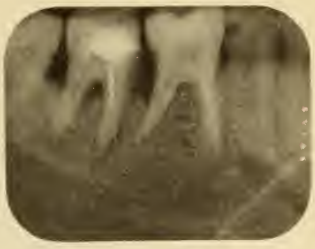


FIG. 150

FIGS. 149 and 150.—Parietal perforations.

foration should be closed before or after the treatment of the root canal depends upon conditions. Usually, asepsis of the canal should be established prior to the closure of the false opening.

The primary aim of treatment should be directed against stopping an existing hemorrhage. Strong caustics must be avoided. The tight plugging of the canal with cotton moistened with a mild antiseptic, *i. e.*, camphorated phenol, etc., and sealing its orifice with temporary stopping for a day or two is usually sufficient. After asepsis of the canal is established in the routine manner the closure of the perforation becomes now imperative. In deep-seated regions in which the opening through the wall cannot be directly viewed, the root canal should be filled with a non-irritating aseptic

material which can be rendered semiliquid by heat at the time of its application. Hard paraffin is most serviceable for this purpose. A suitable compound may be prepared as follows:

Bismuth subnitrate	ʒj (4 gm.)
Hard paraffin (about 130° F., 55° C.)	ʒij (8 gm.)

Heat the paraffin to about 250° F. (120° C.) for half an hour to insure sterility; let it cool to about 150° F. (65° C.) and add the bismuth under constant stirring. Remelt the solidified mixture and stir well to insure a homogeneous union. Pour upon a glass slab and after cooling remove, cut in strips and preserve in a wide-mouth bottle.

Fig. 151

Fig. 152

Figs. 151 and 152.—Interradicular perforations.

Fig. 153.—Broach passing through the foramen into the antrum—not a perforation.

The initial steps for the preparation of the root canal prior to its filling with paraffin are the same as those discussed in the previous chapter. Complete dryness of the canal for this purpose is essential. A cone-shaped piece of the hard paraffin is inserted in the root canal and the heated root dryer is passed along its side opposite the perforation. By a gentle pumping motion the air is expelled and the semiliquid paraffin is coaxed into the canal and the perforation. Care should be exercised not to overheat the paraffin, although it is essential to keep the root dryer fairly warm so as not to chill the compound. A jet of warm air applied during the filling procedure materially assists in keeping the paraffin soft. A suitable sterile gutta-percha cone with its tip cut off may now be

introduced and pressed to place, thereby acting as a core and insuring a more perfect adaptation of the softened paraffin to the irregularities of the canal. The final closure of the canal is the same as in an ordinary canal filling. A roentgenogram of the finished operation will furnish a distinct shadow picture of the bismuth-paraffin root filling and it will indicate whether the perforation has been successfully occluded.

If the perforation is visible it is, of course, more easy to close the aperture. Sometimes a thin bladed burnisher may be inserted between the pericementum and the opening or into the bifurcation of the roots in an interradicular perforation. The thin blade acts as a matrix. Visible perforations are best closed with soft copper amalgam. Should there be any objection to the use of amalgam the opening may be sealed with a suitable piece of metallic foil dipped into a varnish or into thin chloro percha. A specific influence of the respective metals used for such purposes, *i. e.*, platinum, gold, lead, etc., upon the success of the operation aside from their varying degrees of adaptability must be denied. If the perforation is caused by caries the dentin within the vicinity of the false opening should be saturated prior to its occlusion with a 2 per cent silver nitrate solution.

If the perforation is located near the apical region and has not been successfully occluded as demonstrated by a roentgenogram, amputation of the involved root may be performed, provided the general conditions of the tooth justify such a procedure.

III. ROOT CANAL FILLING MATERIAL WHICH HAS PASSED BEYOND THE FORAMEN.

Root-filling material which has been forced into the periapical tissue during an attempt of filling a canal occasionally causes disturbances within this region by irritation and if it is not aseptic it naturally will be the cause of a future infection. Of the cements, the oxychlorid is probably most frequently employed as a root-filling material. If a small quantity is passed above the foramen it is usually followed by more or less painful irritation lasting for about twenty-four hours. Only when large quantities are forced into the periapical tissues are serious consequences to be expected. Root-filling materials containing caustic drugs such as trioximethylen (paraform), etc., should never be employed; they usually cause necrosis when brought in contact with the soft tissues with all the undesirable sequences. Gutta percha in the form of cones or in its various semisolid modifications are frequently pressed into the periapical space during the process of root-filling. As stated, if the material is sterile and the protruding quantity does not

impinge too severely upon the tissues it becomes incapsulated and no ill results are to be expected. Many operators wilfully pass small quantities of gutta percha above the foramen, especially in such cases in which a crater-like depression is present at the apex of the root. The advocates of this procedure claim that the capping of the crater is the only safeguard of having the root canal completely sealed.

Immediate root-canal filling should never be practised in cases in which a pulp has been removed under local anesthesia; at least twenty-four hours should be allowed for the return of normal sensation of the disturbed periapical tissues.